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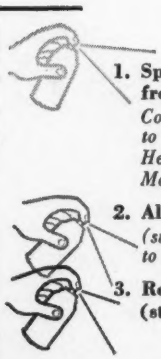

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THE
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BETTER SURGERY

PHILIP B. PRICE, M.D.*

Salt Lake City, Utah

In the long and sometimes splendid history of surgery, there has been for solid achievement no period comparable to the present. A surgical textbook published 20 years ago now appears strangely deficient and obsolete. Those of us who graduated before that time have had to discard much of what we were taught in medical school and in our hospital training. Old concepts have been abandoned as new vistas of knowledge have opened.

It has been an exciting time in which to live, for each year has brought significant new advances. Antibiotics and control of infections; new anesthetic agents and better administration of anesthesia by a new generation of pharmacologically-minded anesthesiologists; blood and plasma transfusions and the virtual disappearance of surgical shock; knowledge of fluid and electrolyte problems and improved preoperative and postoperative care; rapid development of pulmonary and cardiac and vascular surgery, and the surgery of childhood; sustained nutrition, early ambulation, and elimination of the old, long period of postoperative debility; anticoagulant therapy; surgery of the adrenals and autonomic nervous system—all of these and many more that could be mentioned are new or revised chapters in surgery.

We all may take pride in these achievements—pride that our medical profession has made them—pride that the rank and file of American surgeons have kept so well abreast of this new scientific knowledge, and have been so successful in putting the new principles and technics into practice—pride that so large a contribution thus has been made to the welfare of mankind. Probably no other profession has progressed so rapidly, and in no other field has the entire membership participated so fully in a forward movement requiring individual teachability of a high order, and often the acquisition of new skills.

From the Department of Surgery, University of Utah College of Medicine, Salt Lake City, Utah.

Presidential address presented during the annual assembly of The Southwestern Surgical Congress, Oklahoma City, Oklahoma, Sept. 20–22, 1954.

* President, The Southwestern Surgical Congress.

Nevertheless—or should I say *therefore?*—present day surgeons are in danger of falling into complacency. Through pride and satisfaction in our new powers over disease and the added safety of major surgical procedures—intoxicated by the superabundance of potent drugs placed at our disposal—we are apt to be blind to grave deficiencies that still exist in our knowledge, and to serious errors that have developed along with the improvements. *Tares* have grown as well as *wheat*, but unlike the man in the parable, we dare not defer to some future date the task of weeding them out. It is something we must do ourselves. The *tares* will not die out spontaneously, nor can anyone else do the weeding for us. This is a matter that should concern every surgeon who loves his profession; is proud of its accomplishments; and earnestly wishes to see it become better and better.

Let me mention some of the more obvious and correctable defects:

1. *The Overuse and Misuse of Drugs.* Surgeons are not alone at fault here. We all use too many therapeutic agents irrationally. It is the line of least resistance. We make easy targets for glib and persuasive drug salesmen and attractively packaged free samples. We are prone to give drugs without full knowledge, sometimes with very scanty knowledge, of their pharmacological action. Multiplicity of drugs in treatment is the order of the day.

An outstanding example of this abuse is the indiscriminate employment of antibiotics. A large percentage of all drugs sold on the American market today are antibiotics. They are used universally, with and without good reason. They are given uselessly for the common cold. They frequently are administered irrationally for various sorts of viral infections. They are prescribed before the patient is examined or a diagnosis is made, as a sort of unscientific therapeutic shortcut. They are employed unreasonably for prevention and treatment of all sorts of surgical complications. As a result the increase in incidence of drug-resistant bacterial infections has increased alarmingly.

The public has learned to demand that sort of medication. If *this* antibiotic does not give the desired results, try *that* one. If *this* doctor will not prescribe the *miracle drug*, get *one* who will. I know of a hospital where, as a matter of policy, every surgical patient receives antibiotics before operation and every day thereafter for at least a week, even the simple clean cases. Nor are university hospitals above criticism in this regard. A random check of our own teaching hospital one day last week revealed that antibiotics were being given to 49 per cent of the surgical patients on the wards; to 28 per cent of the medical in-patients, and to 67 per cent of the pediatric patients.

Pleas for more restrained and rational use of antibiotics, for more frequent identification of the offending organisms and employment of sensitivity tests have been made by Frank Meleney, Edwin Pulaski, and other leaders in this field. Drugfastness has become a major nation-wide problem.

For the sake of better and more scientific medicine and surgery, the excessive use of drugs of all sorts without clear and specific indication is a trend that needs reversal. A great many of the newer drugs are powerful agents, potentially dangerous, and capable of causing profound disturbances of the patient's physi-

ology and biochemistry. They should be used with knowledge and caution. An editorial in the last (September 1954) issue of *The American Surgeon* cites Efocaine as "another example of the use of drug therapy in human beings producing disastrous results before adequate basic anatomic, biochemic, physiologic, and pharmacologic studies have been completed in the experimental animal."

2. *Errors Related to Anesthesia.* Because anesthesia is inextricably tied to surgery, and necessarily remains in part the responsibility of the surgeon, I shall make bold to point out some defects in this field that need correction also. No one will deny that great progress has been made in anesthesiology in recent years, or that administration of anesthesia is on the whole far better than it used to be. Admittedly this improvement has contributed largely to the expansion of certain fields of surgery and the safety of major surgery in general. Nevertheless, in the interest of further improvement in surgery, certain trends in present day anesthesiology need correction.

For one thing, an unfortunate spirit of independence and even antagonism has developed, at least in some quarters, between anesthesiologists and surgeons. Now the surgeon is at fault if he treats his colleague—the trained anesthesiologist—as autocratically as he was wont to treat nurse anesthetists. On the other hand, it is equally culpable for the anesthesiologist, in his pride in the new position and importance of his field, to take a high-handed attitude. He has no right to say to his surgical colleague, "You pay attention to your operating and I'll handle the anesthesia." The surgeon cannot legally or morally relinquish all responsibility for the anesthesia, even if he wishes to. Nor can the patient be divided up in that manner. He is a single unit of interrelated and complex physiologic and chemical systems and activities. The ideal is cooperation—surgeon and anesthesiologist working together as partners in a single task, agreeing on matters of preoperative and postoperative care, scheduling of the operation, choice of anesthetic agents and supplementary medication, and the administration of blood and fluids. The surgeon should keep the anesthesiologist appraised of the operative findings and procedures and the condition of the patient as seen from his vantage point, and the anesthesiologist in turn should keep the surgeon informed regarding the agents which he is administering and his estimate of the patient's condition.

In my opinion—and in the opinion of many surgeons and physicians—operative patients are being subjected too often at the present time to tracheal intubation. It is not an atraumatic procedure. We are seeing many postanesthesia patients with rhinitis, laryngitis and tracheitis for which they receive antibiotics with attendant risks of drug reaction and drugfast infection. I dare say that in a large majority of ordinary operative patients, perfectly adequate airway can be maintained without intubation. Skillful use of the oral airway and maintenance of an air passage simply by holding up the chin seem to be forgotten arts. Although artificial respiration is essential in thoracic surgery and under certain other special conditions, I submit that ordinarily the anesthesiologist using "controlled" respiration cannot hope to do as well as can the complicated, delicately

adjusted, automatic mechanism of the body in regulating the respiration and circulation, or in perfectly adjusting oxygenation and the chemistries involved to the constantly changing needs of the unconscious patient.

I also am one of those who believe that multiplicity of drugs employed in present day anesthesiology is a trend that needs amending. Most of them are powerful agents. It is not reasonable to assume that by giving small does of a succession of drugs one can gain all the desired pharmacologic actions and none of the side effects. Few anesthesiologists would claim to be experts in the pharmacology of these agents taken singly, and none can predict the total effect on any particular patient when they are given one after another in rapid succession. I have recorded elsewhere an instance in which I have observed a patient receive scopolamine, morphine, atrophine, sodium pentothal, cyclopropane, oxygen, nitrous oxide, neosynephrine, curare, demerol, and quinidine, all within a few hours. That probably is not an unusual experience. Sometimes these drugs are given without consultation with, and even without the knowledge of, the surgeon. The current high rate of operative and postoperative complications, I believe, is related to this machine gun sort of therapy. Henry Beecher and his co-workers, after a 5 year study in 10 representative hospitals across the country, concluded that curare and other muscle relaxants, so freely employed nowadays, must be credited with causing many "anesthetic deaths". And yet curare is only one of the potentially dangerous adjuvants to anesthesia in common use at the present time.

3. *Excessive Use of Intravenous Fluid and Blood.* Intravenous salt solution and glucose solution have an important place in proper preoperative, operative, and postoperative management of patients, but it is a mistake to give indeterminate amounts routinely. Knowledge and good judgment are required to meet the needs of any given patient in this regard; but, if one must err, let it be on the side of dehydration rather than over-hydration. Perhaps we have been unwise in abandoning altogether the relatively safe measures of proctoclysis and hypodermoclysis, and in limiting oral intake too strictly, both before and after operation. American visitors to England and Scotland are impressed with how few patients on the surgical services are receiving intravenous fluids, in contrast to many American hospitals where wards are often veritable forests of infusion stands, bottles, and tubes.

It now is customary, and has become almost fashionable, to give 1 or more units of blood during all major operations, whether it is needed or not. This responsibility often is delegated to the anesthetist who is guided solely by the patient's blood pressure. Transfusions are safer than they used to be, but they still carry some risk—risk of severe reactions, renal shutdown, cardiovascular accidents, homologous serum hepatitis, transmission of disease, and so on. Kilduffe and DeBakey in their book report a collected series of about 43,000 transfusions with 80 hemolytic reactions and 45 deaths. It has been estimated that there are now more deaths in the United States attributable to transfusions than to appendicitis.

There also is the more insidious danger than the surgeon and his assistants

may become sloppy surgical technicians, careless of hemostasis since lost blood can be so readily replaced. Meticulous hemostasis may be troublesome to practice, but it does enable the patient to keep his own blood. Most operations of small or moderate size *can* be done with relatively small amounts of blood loss, no more than the average patient can tolerate readily. These patients often do better than those who are permitted to bleed considerably and then are transfused.

When significant amounts of blood or serum are lost during and following operation, these vital fluids must be replaced, of course. Ideally they should be administered as the losses occur, and in approximately those amounts, so that the patient's blood volume remains virtually unchanged throughout the whole episode. The hypothesis advanced a few years ago that chronically ill and debilitated patients need large amounts of blood to restore an arbitrary *ideal* blood volume has some basis of truth, but accepted uncritically that teaching has led to excessive use of blood.

4. *Neglect of Fundamental Principles of Surgery.* Certain fundamental principles of surgery, forged out of experience, have been bequeathed to us as a priceless heritage from preceding generations of master surgeons. These principles are in danger of being discarded. That would be retrogression. Careful hemostasis, gentle handling of tissues, perfect asepsis, anatomic repair, and restoration of physiologic function are as truly the marks of good surgery today as they ever were. However, readily available transfusions have made operators careless about hemostasis; antibiotics have made them less careful about sepsis; good nutrition and relatively good physical status have helped to conceal the ill effects of rough treatment of tissues and imperfect repair; and control of fluid and electrolyte balances and the use of influential drugs have tended to obscure postoperative physiologic dysfunction. Yet, in the long run, ill effects become apparent and patients suffer when these basic tenets of good surgery are ignored.

5. *Unjustifiable Surgery.* One of the shocks in store for well-trained, idealistic young surgeons starting in practice is the discovery that much needless, ill-considered surgery is being done, often by perfectly ethical, well-intentioned doctors. It is so the country over. All of us have observed innumerable instances of this misuse of surgery. There are operators who, failing to find an expected lesion, or sufficient pathology to account for the symptoms, think that they must do something anyhow, so they take out a normal uterus, or the ovaries, or an innocuous gallbladder, or do a gastroenterostomy, or resect the stomach without adequate reason. One sees breasts removed simply because they are a little lumpy; tonsils removed only because they are large; uteri suspended in the vain hope that infertility or backache will be cured thereby. Some doctors repair innocent umbilical hernias in infants, or attempt to bring down undescended testes in early childhood, not realizing that nature, given time, may and often does take care of those developmental problems spontaneously. Zealous operators sometimes attempt radical surgery on manifestly inoperable malignancies, until hard experience teaches them to be content with palliative procedures or no

operation at all. In case of incurable breast carcinoma, Haagensen and Stout have shown that the less surgery done, the longer the patients survive.

No arbitrary standards can be set up to indicate what constitutes justifiable surgery in every instance. Each surgeon must be guided by his own best judgment and a good conscience. The safe rule is the Golden Rule. What would I want done to me, surgically, if I were the patient?

6. *Unworthy Motivation.* Finally, there is an unhappy set of abuses arising from distorted scales of values, from failure to place the patient's welfare above all other considerations. Perhaps none of us is entirely free from fault in this regard. We know better, but our frail human nature betrays us. Saint Paul's lament, "The good that I would I do not, but the evil which I would not that I do," finds an echo in many an honest surgeon's heart.

We rightly condemn the man who does unnecessary surgery for the sake of income, but we are not blameless ourselves if we let consideration of fees or lack of fees influence our judgment or the care we accord a patient. Avarice and selfish pride are the base motives which underlie ghost surgery, fee splitting, peripatetic surgery, and reluctance to seek consultations or to refer patients. Most of us endorse the position of the American College of Surgeons and other groups in denouncing such practices, but at the same time we would do well to examine our own secret motives to see whether we ourselves are free from all taint of avarice and false pride in our dealings with patients. An insatiable desire for more income and prestige leads some surgeons to bend every effort to expand and protect their practices until eventually they overload themselves with work to the detriment of the quality of their service. Such men would live longer and more healthily and more happily if they would add a little leaven of generosity to their make-up, and turn over to struggling young surgeons in their community some of their surplus cases, or the industrial contracts they have needlessly clung onto.

Thoughtlessness, sheer laziness, and inconvenience are sometimes responsible for wrong or unnecessary surgery. How frequently, for example, one encounters the sorry spectacle of a patient operated upon for *hemorrhoids* who really had a carcinoma of the rectum that went undiagnosed for want of a simple digital or proctoscopic examination. Exploratory operation has a legitimate and valuable place in surgery, and in certain acute conditions it becomes mandatory to operate immediately before a positive diagnosis can be reached, but, unless we are very careful, that sort of surgery can be abused. It is unfortunate, and sometimes tragic, when patients with pneumonia, or coronary disease, or hepatitis, or acute pancreatitis, or gastroenteritis, or acute gonorrheal salpingitis, or acute nephritis are subjected unnecessarily to operation, especially when more careful examination and a little more forethought might have saved them that discomfort, risk and expense. On the other hand, there are sins of omission in surgery that are equally reprehensible. To operate on an *acute abdomen*, find and remove a virtually normal appendix, and then close up without search for diverticulitis, perforated ulcer, intestinal strangulation, or other explanation of the patient's condition is inexcusable. To procrastinate or refuse to operate at all in the face of

clear indications is culpable, especially if the refusal is motivated by indolence or inconvenience, or fear that an unsuccessful outcome might jeopardize the surgeon's personal reputation.

To call a case an *emergency* when it really is not one, just to secure a bed in a crowded hospital or a place on a tight operating room schedule, is dishonest and essentially selfish, and really is not fair to the patient.

Clinical experimentation is permissible and often desirable, but not at the cost of a patient's wellbeing. It is popularly believed that university hospitals are places where patients are experimented upon freely. On the contrary, it usually is the staffs of those hospitals that insist most strongly that all clinical experimentation should be carefully planned and executed, usually with the consent of the patient, with such controls as can be devised, and often only after the probable value and safety of the procedure has been demonstrated in the laboratory. Scientific curiosity or showmanship should never be placed above the welfare of the patient. Haphazard experiments, with scalpel or drugs, are rightly condemned.

Ideals. Most of us are idealists. Lingering traces of that charming, nebulous quality of youth cling to all but the cynics, and perhaps secretly to them also. We instinctively feel a need for something better and nobler than ordinary to aspire to, even though our reach may exceed our grasp.

May I suggest a few principles that seem to me to be worthy goals for a surgeon? They are not new thoughts, of course. Maybe some of you can phrase them better than I have.

Treat people, not diseases alone.

View each patient as an individual of inestimable worth. Your responsibility for his care shall be held a sacred trust, and his welfare shall come before any good or convenience to yourself.

Strive to do every patient more good than harm.

Do not administer drugs unnecessarily or ignorantly.

Make each operation a feat of fine workmanship.

Be humble enough to seek consultation whenever it is indicated, and unselfish enough to refer patients when that is to their best interest.

Do not perform any surgery, nor remove any organ or part, without justifiable indications therefor.

Endeavor to make surgery as safe as possible for every patient; on the other hand, should hazardous surgery be indicated, do not out of self interest deny to a patient whatever chance of benefit the operation may offer.

Let him who is so inclined label such sentiments "platitudes" or "counsels of perfection." We need counsels of perfection. We are better men for paying heed to them.

CONCLUSION

In these reflections I hope I have managed to avoid the unhappy role of a scold; instead, I have aimed at a combination of appreciation, realism, and constructive criticism. I am grateful for the wealth of new therapeutic agents

placed at our disposal; but I note that they are being overused and sometimes misused, and I join others in urging more restraint and rationality in their employment. I admire and have benefitted from recent progress in anesthesia; at the same time I am disturbed by imperfect cooperation between surgeons and anesthesiologists; by the excessive use of tracheal intubation, and by the too liberal use of supplementary medication; and I urge concerted efforts to correct those defects. No one appreciates more than I do the contribution made to surgery by blood transfusions and blood substitutes, but I cannot help observing that this supportive therapy often is being employed excessively and unwisely; hence a plea for clearer understanding of specific indications for its use. I deplore the neglect of time-honored, experience-tested, fundamental principles of good surgery. These basic tenets need to be taught and practiced more conscientiously. The increasing safety of surgical procedures is a priceless boon, but it also has permitted the performance of much unnecessary surgery. Although this abuse is being opposed by organizations, and by groups of high-minded surgeons and pathologists, with some success, hope for betterment lies chiefly in the innate idealism of individual surgeons. The most baffling and disturbing defects in the practice of surgery today, however, come from wrong motivation. This is a particularly difficult problem because faulty human nature is involved and none of us is entirely blameless. Here is a matter that deserves serious study. Pious lectures to undergraduate students on medical ethics is not the answer. What is needed is a rebirth of idealism and altruism in the practice of surgery.

In a word, the *art of surgery* has not kept pace with the *science of surgery*. Progress in the science of surgery has resulted largely from the work of a few brilliant men carrying out researches on wards and in experimental laboratories. The rest of us are their followers and disciples. But progress in the art of surgery cannot be left to any small group. It will come, if at all, by the combined and whole-hearted efforts of the entire body of surgeons. Here is a problem that calls for constructive thinking as well as idealism. It is a responsibility that you and I cannot avoid. It is an opportunity that we should welcome. Perhaps in no other way can we, as individuals and as a group, contribute so much to the betterment of surgery.

THE DIAGNOSIS AND TREATMENT OF HEAD INJURIES

A. C. Lisle, Jr., M.D.

Oklahoma City, Oklahoma

The staggering incidence of head injuries makes it imperative that the management of such cases remain primarily the responsibility of the general practitioner and, especially, the general surgeon. This is significantly shown here in the United States where approximately 35 per cent of all accidental deaths and 60 per cent of automobile deaths are due to head injuries. It is, therefore, necessary that we be able to recognize and treat these patients, noting the danger signs and complications warranting neurosurgical consultation. The purpose of this paper is to outline briefly the essential considerations of diagnosis and treatment of head injuries.

INITIAL BASE LINE

To manage head injuries properly, it is essential to create an initial base line[10] from which to evaluate the patient's future course. This consists of the following in their order of importance:

1. The state of consciousness: When the physician first sees the patient, he should determine whether the injured person is only dazed, stuporous or totally unresponsive, so he will be aware of further changes of consciousness.

2. Changes of the pupils: He should observe the size, equality and reaction of the pupils for future reference.

a. Contracted, small pupils are seen in severe brain stem injuries and denote a bad prognosis. It is well to know whether or not the patient has had morphine which also constricts the pupils.

b. Dilated, fixed pupils are seen in profound brain injuries. This is almost invariably a grave and terminal sign.

c. Unequal pupils: The dilated pupil usually is without reaction to light and suggests an ipsilateral, extradural hematoma, temporal lobe contusion or orbital injury.

3. The alteration of the vital signs: This includes the pulse, blood pressure, respiration and temperature. Increasing intracranial pressure is recognized by a progressive lowering of the pulse and a paradoxical rise of the blood pressure. Early hyperthermia, not arising from some pulmonary or other complication, is directly proportional to the severity of the brain injury. Slow, labored respiration, i.e., Biot's respiration, is a grave sign, indicating failure of the medulla and impending death due to respiratory paralysis. Serial recording of these vital signs is important to detect the changes that may be occurring.

4. The presence or absence of paralysis: Immediate hemiparesis usually occurs with severe local cerebral contusion; but paralysis occurring several hours or

From the Department of Surgery, University of Oklahoma School of Medicine.
Presented during the Oklahoma City assembly of The Southwestern Surgical Congress, Sept. 20–22, 1954, Oklahoma City, Okla.

days later suggests subdural hematoma. Hemiplegia is detected readily in the drowsy patient. In the stuporous patient, it may be evident by painfully stimulating him and noting the comparative reaction of his right and left sides. In the comatosed patient, the paralyzed side of his face tends to puff out with expiration. Absence of the reflexes may be noted on the side of the paralysis. Total areflexia is an ominous sign. A stiff neck shortly after injury suggests a fractured cervical spine but after the first 24 hours, it probably is due to irritation of the meninges by red cells in the spinal fluid.

CONCUSSION

Concussion is a term used loosely by physicians, attorneys and the laity; but is correctly defined as a transient loss of consciousness following trauma, not exceeding 10 minutes' time, and without gross injury to the brain. Most patients whom we see with head injuries have more than a concussion; namely, cerebral contusion manifested by unconsciousness for longer than 10 minutes, amnesia, headaches, dizziness and, occasionally, convulsive seizures.

Headaches, dizziness, nervousness and impairment of memory erroneously have been called postconcussion syndrome. Extensive study has shown there is no correlation between the persistence of these symptoms and the severity of the head injury. Many factors play a part, such as the patient's pretraumatic personality, social factors and pending litigation. Early ambulation will reduce the frequency of these complaints. Serious mental symptoms are accompanied by cerebral atrophy which can be demonstrated by pneumoencephalography.

GENERAL MANAGEMENT

Careful and repeated observation of the patient is the basis of good management. Conservative treatment as well as preoperative and postoperative care of surgically treated patients consist of the following:

1. *Maintenance of an Airway:* This is by far the most important single phase of the treatment. Anoxia produces irreversible changes of the brain within 13 seconds; therefore, it is extremely important to maintain a good airway in the unconscious patient. Anoxia enhances cerebral edema. Place the patient on his side so secretions can run from his nose and mouth. One should not hesitate a moment in making a tracheostomy, if necessary. Oxygen should be administered; if he is unconscious, he should be turned from side to side every two hours.

2. *Management of Shock:* The initial administration of intravenous fluids, plasma or blood and appropriate vasopressor drug should be followed immediately by seeking the cause of shock. Head injuries alone with the exception of extensive scalp laceration rarely are accompanied by shock; therefore, a careful search must be made for a severe injury of an extremity, intra-abdominal hemorrhage, hemothorax or fractured spine.

3. *Maintenance of Fluid Balance and Nutrition:* The average adult should receive approximately 2500 cc. of fluid daily. This may be given by vein or subcutaneously during the first three days. Then, if he still is unable to take liquids by mouth, a stomach tube may be inserted for high caloric gavage feedings. The use of hypertonic solutions and fluid restriction has been discontinued by prac-

tically all neurosurgeons. Such a practice is not only of no value, but often harmful. There is abundant evidence[6, 15] that fluid can be withdrawn from the brain with such solutions, but this is very transient and often is followed by even more edema than was originally present.

4. *Management of Agitation, Restlessness and Pain:* Mild restlessness in the confused or stuporous patient usually can be handled by placing side rails on the bed. Overly solicitous relatives tend to aggravate the agitation, and when restlessness is extreme, sedation must be used. Paraldehyde is the drug of choice; however, barbiturates may be used, if the respiration is satisfactory. In some patients, pain alone is responsible for their agitation, such as a distended bladder. Codeine is a good analgesic. Morphine should not be used because of its depressing effect on the respiratory centers which may already be in some jeopardy.

5. *Control of Hyperthermia:* Hyperthermia is rather frequent in brain injuries because the hypothalmus is so vulnerable to trauma. The higher the temperature, usually the more severe the brain injury. Vigorous efforts to help the patient eliminate heat are necessary. Removal of all clothing and covering, sponging, ice bags, oxygen and repeated large doses of dissolved aspirin by rectum or gavage tubes usually suffice. In more resistant cases, cover the body with iced towels and cool with an electric fan. Ice water enemas and a rubber mattress filled with ice water may help.

6. *Chemotherapy:* Profoundly unconscious patients should receive some form of chemotherapy to reduce the risk of complicating pneumonia or urinary tract infection. This is true particularly in open wounds and basilar skull fractures with spinal fluid leakage. I favor the use of penicillin and sulfadiazine or gantrisin. The sulfa drugs will readily pass the blood-brain barrier.

7. *Early Ambulation:* This is very important. As soon as the patient is conscious enough to sit up, he should be allowed to do so. He should be urged to be up and about on the first day, if possible. This reduces venous congestion of the brain; helps reduce edema and lowers the incidence of post-traumatic neuroses.

8. *Miscellaneous:*

a. Indwelling catheter: This is helpful to overcome restlessness in the stuporous patient and to lessen the likelihood of decubiti by keeping the bed dry. Frequent irrigations are desirable.

b. Atropine sulfate[8, 14] in large doses, e.g., 2 mg., for the average adult has a beneficial effect upon the parasympathetic responses, i.e., hypotension, diaphoresis and increased gastrointestinal motility that accompany cerebral trauma. It blocks the muscarinic responses of acetylcholine which apparently is liberated from injured brain tissue.

c. Cortisone has been used in severe head injuries, giving 300 mg. the first day and gradually decreasing the dosage during the ensuing week.

SURGICAL TREATMENT

Only about 10 per cent of head injuries require surgery. The most important single indication for seeking neurosurgical consultation is the progressive impairment of consciousness.

Skull Fractures: Undue emphasis frequently is assigned to skull fractures, for-

getting the more significant injury of the brain. There is no correlation between these injuries. One may have a skull fracture without brain damage or, on the other hand, have a fatal brain injury without a fracture of the skull. It is a mistake to rush every patient to the radiology department for skull films. This should be deferred until his general condition permits. In any event, roentgenograms seldom influence the treatment of the patient.

1. *Linear Fractures of the Skull:* Linear fractures require no special treatment and the patient may be up and about from the very first day.

2. *Depressed Skull Fractures:* These are seldom emergencies. One usually may wait several days to allow the patient to stabilize before undertaking surgical elevation of the depressed fragment. An inviolable rule is that all depressed skull fractures should be elevated. This lessens the incidence of post-traumatic seizures.

3. *Compound Fractures and Compound Depressed Skull Fractures:* These are emergencies. A pressure bandage may be applied to control bleeding. When the patient's condition permits, the scalp should be shaved widely about the wound, shaving away from the edges. After the scalp has been carefully cleansed with soap and water, the wound is irrigated copiously with warm sterile physiologic sodium chloride solution. Local anesthetic of 0.5 per cent novocaine is injected around the wound. If the patient is combative, intravenous pentothal may be used. Careful debridement is done, starting with the scalp and proceeding to the periosteum, skull, dura and brain. Hemostasis is secured and careful dural closure is made or a fascial graft is inserted. The galea and skin each are closed with a layer of interrupted silk sutures. Cranioplasty is delayed approximately six months to obviate infection which would require removal of the metallic or acrylic plate or graft.

Basilar skull fractures are most significant when they extend into the ear, sinuses, nose or roof of the orbit, opening portals of entry for infection into the intracranial cavity. These fractures usually are accompanied by leakage of blood-tinged cerebrospinal fluid from the ear or nose. Roentgenographic demonstration of these fractures is difficult. Treatment consists of strict bed rest with the head of the bed elevated. There should be no irrigating or packing of the involved nostril or ear. Penicillin and sulfadiazine or gantrisin should be given for five days after leakage stops or until the patient has been afebrile five days. If the otorrhea or rhinorrhea persists longer than one week, then craniotomy, with repair of the tear in the dura, should be done. These patients should not have spinal tap as it might tend to suck infected material into the head by lowering the intracranial pressure. Meningitis and brain abscess occasionally complicate this type of fracture.

SPINAL PUNCTURES

The indications for lumbar puncture in head injuries are:

1. The diagnosis of associated subarachnoid hemorrhage.
2. The diagnosis of complicating meningitis.
3. Severe, persistent headache may be relieved in a few people.

The routine used of lumbar tap in head injuries is unjustifiable. The intra-

cranial pressure can be reduced temporarily[5]; but how long this lowered pressure persists is debatable. Spinal tap is hazardous in increased intracranial pressure because it may precipitate herniation of the temporal lobe into the incisura of the tentorium. Such herniation[9] may complicate cerebral contusion, cerebral edema or intracranial hematomas. This is a serious complication and may be accompanied by decerebrate (extensor) rigidity and medullary failure due to the brain stem compression. Tentorial herniation has been treated by manual reduction of the herniated temporal lobe,[12] division of the tentorium and removal of the contused and pulped brain tissue.

INTRACRANIAL HEMORRHAGE

1. *Subarachnoid Hemorrhage:* This is rather frequent in head injuries. Few symptoms occur within the first 24 hours. Then the red blood cells break down and release their hemoglobin into the spinal fluid. This substance produces meningeal irritation manifested by headache, fever, irritability, photophobia, stiff neck, nausea and vomiting. Diagnosis by lumbar puncture differentiates this from bacterial meningitis. Repeated lumbar taps are ineffective in removal of red blood cells from the spinal fluid as shown by Sprong.[11] Conservative, general management usually suffices for these patients.

2. *Intracerebral Hemorrhage and Acute Subdural Hematoma:* These are seen in severe injuries, especially with lacerations of the brain. There is no classical description for these two conditions. The symptoms are similar to those of cerebral contusion and often diagnosed erroneously. Bilateral burr holes are indicated whenever there is progressive impairment of consciousness or other signs to indicate increasing intracranial pressure and neurologic dysfunction. Although the hematoma is evacuated, the mortality rate is near 95 per cent and the sequelae are severe.

3. *Extradural Hemorrhage:* An extradural hemorrhage arises usually from a torn middle meningeal artery or, occasionally, a dural sinus. Epidural hemorrhage occurs in about 3 per cent of acute head injuries.[16] The typical epidural clot produces a classical syndrome. The patient is momentarily unconscious from a blow on the head, regains consciousness and is rational (lucid interval); only to lapse into a stupor coma again. The pulse becomes slow, the blood pressure rises and the respiration becomes slow and labored (Biot's respiration). There may be localized swelling of the scalp. Usually the ipsilateral pupil is dilated due to compression of the oculomotor nerve by herniation of the temporal lobe through the incisura of the tentorium. The opposite leg and arm are paretic. Terminally, extensor rigidity may occur. If roentgenograms of the skull have been made, a linear fracture may be seen in the temporal bone, crossing the groove made by the middle miningeal artery. Because the bleeding is of arterial origin, the symptoms develop rapidly, necessitating prompt recognition and surgery. A burr hole is made in the temporal bone; the jelly-like hematoma is evacuated and the bleeding from the meningeal artery is controlled. The mortality rate is about 50 per cent and the sequelae are usually mild.

4. *Chronic Subdural Hematoma:* This is the most common type of hematoma.

These usually arise from one of the bridging cortical veins extending from the surface of the brain to the saggital sinus. This has been clearly demonstrated experimentally by Pudenz and Sheldon.[7] These hematomas occur in infants, people past middle age and, especially in alcoholic addicts. About one-third are found bilaterally. The injury may be severe or trivial and soon forgotten; only to begin showing symptoms days or weeks later. Headaches and drowsiness which tend to wax and wane are the outstanding symptoms. Hemiplegia and convulsions are frequent. Papilledema is present in about half of these patients. The spinal fluid pressure usually is elevated. It may be clear or xanthochromic and frequently contains an elevated protein content. Roentgenograms of the skull may show a linear fracture, shift of the pineal gland and, rarely, a case of long standing may show calcification of the hematoma capsule. Bilateral burr holes are made in the upper temporal region. Should no clot be found, additional openings are made or ventriculography may be resorted to in localizing the hematoma.[1] The presence of a subdural hematoma produces a blue discoloration of the overlying dura. Satisfactory drainage of the liquid hematoma, as first described by Fleming and Jones,[2] usually can be done through a burr hole by carefully irrigating the subdural space with sterile warm physiologic sodium chloride solution. The incisions are closed without drainage with interrupted silk sutures in the galea and skin. Occasionally, it is necessary to reflect a bone flap, if the hematoma is solid clots. The blood which accumulates in the subdural space, after injury, disintegrates within a few days and fibroblasts form a surrounding capsule or sac. This sac and its liquid contents gradually swell from the increased osmotic action of the distintegrating red blood cells as described by Gardner.[3] The postoperative care of these patients is not a real problem and the average patient improves the first day or so.

Differential diagnosis from brain abscess and tumor is difficult depending primarily on the history; i.e., trauma and sinus infection. A subdural hygroma consists of a loculated collection of light xanthochromic fluid in the subdural space; probably due to a small tear in the arachnoid allowing cerebrospinal fluid to leak into the subdural space where it becomes trapped. The symptoms, diagnosis and treatment are the same as in subdural hematomas.

A subdural hematoma in infants is a problem in differentiation from hydrocephalus. These babies are fretful, irritable, eat poorly and gain slowly. They frequently have an enlarging head and convulsions. Diagnosis is made by puncture through the anterior fontanel with removal of xanthochromic fluid from one or both subdural spaces. Babies with hydrocephalus yield clear fluid by fontanel tap at a depth of approximately 2 centimeters. Definitive treatment is the same as in adults although Ingraham[4] stresses removal of the hematoma capsule because the growing brain may be retarded in its development, if it is not removed.

Post-traumatic Epilepsy: Convulsive seizures complicating head injuries may develop several weeks or years after injury,[13] but in the majority of patients within six months. The frequency of seizures in closed head injuries is approximately 3 per cent; whereas, open wounds of the head are followed by convulsions

in approximately 15 per cent. Surgical treatment of the complication is reserved for the patients not effectively controlled by anti-convulsant medication and who have focal seizures and/or electroencephalographic changes.

SUMMARY

The staggering incidence of head injuries makes it imperative that the management of such cases remain primarily the responsibility of the general practitioner and, especially, the general surgeon.

The salient diagnostic considerations of different types of head injuries, including concussion, skull fracture and hematomas are discussed. Particular emphasis is given to the general management of these injuries. The indications for surgical treatments are reviewed; noting particularly the danger signs and complications.

REFERENCES

1. Evans, J. P.: Acute Head Injury, Springfield, Illinois, Charles C Thomas, 1950.
2. Fleming, H. W., and Jones, O. W., Jr.: Chronic subdural hematoma; simple drainage as method of treatment; report of 8 cases, Surg., Gynec. & Obst., *54:* 81 (Jan.) 1932.
3. Gardner, W. J.: Traumatic subdural hematoma, with particular reference to latent interval, Arch. Neurol. & Psychiat. *27:* 847 (April) 1932.
4. Ingraham, F. D., and Matson, D. D.: Subdural hematoma in infancy, J. Pediat. *24:* 1 (Jan.) 1944.
5. Mock, H. E., and Mock, H. E., Jr.: Management of skull fractures and brain injuries, J. A. M. A. *120:* 498 (Oct. 17) 1942.
6. Musserman, J. H.: Effects of intravenous administration of hypertonic solution of sucrose with special reference to cerebrospinal fluid pressure, Bull. Johns Hopkins Hospital *57:* 12, 1935.
7. Pudenz, R. H., and Sheldon, C. H.: Lucite calvorium-a method for direct observation of the brain; cranial trauma and brain movement, J. Neurosurg. *3:* 487 (Nov.) 1946.
8. Ruge, D.: Use of cholinergic blocking agents in the treatment of craniocerebral injuries, J. Neurosurg. *9:* 77 (Jan.) 1954.
9. Scheinker, I. M.: Transtentorial herniation of brain stem, Arch. Neurol & Psychiat., *53:* 289 (April) 1945.
10. Segerberg, L. H., and Spurling, R. G.: Acute craniocerebral trauma; essential considerations of diagnosis and treatment, J. A. M. A. *141:* 371 (Oct. 8) 1949.
11. Sprong, W.: Disappearance of blood from cerebrospinal fluid in traumatic subarachnoid hemorrhage; ineffectiveness of repeated lumbar punctures, Surg., Gynec. & Obst. *58:* 705 (April) 1934.
12. Vincent, C., LeBeau, J., and Guiat, G.: Brain edema in neurosurgery, Proc. Reg. Soc. Med. *40:* 689 (Oct.) 1947.
13. Walker, A. E.: Posttraumatic Epilepsy, Springfield, Illinois, Charles C Thomas, 1949.
14. Ward, A. A., Jr.: Atropine in treatment of closed head injury, J. Neurosurg. *7:* 398 (Sept.) 1950.
15. Weed, L. H., and McKibbens, P. S.: Pressure changes in the cerebrospinal fluid following intravenous injection of solution of various concentrations, Am. J. Physio. *48:* 512 (May) 1919.
16. Woodhall, B., Devine, J. W., Jr., and Hart, D.: Homolateral dilatation of pupil, homolateral paresis and bilateral muscular rigidity in diagnosis of extradural hemorrhage, Surg., Gynec. & Obst. *72:* 391 (Feb.) 1941.

INJURIES TO THE LOW BACK

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The problem of the injured low back has perplexed every physician many times. This often involves the difficulty of determining whether or not an injury has taken place and then evaluating the severity of such an injury. To emphasize the difficulties encountered in solving low back problems, one only has to review its complex anatomy. As Watson-Jones[5] has pointed out, there is a complex anatomic system of more than 20 joints in the lower back with innumerable ligaments, muscles and aponeuroses, all of which are arranged in a complex manner. Each of these is susceptible to the same injuries as the joints and ligaments in the extremities. However, the joints and other structures in the back are so deeply situated and are so closely related that it is not possible to examine them as minutely as is possible with a superficial joint. Furthermore, muscle spasm and loss of back motion may be present in injuries to any one or all of the structures in the back. Additional difficulties arise from the complexity of the nerve supply. All of the posterior muscles, ligaments, fasciae and joints are supplied by the posterior primary divisions of the lumbar and sacral nerves. Steindler has demonstrated that pain arising from sprained muscles, ligaments and joints may be referred down the sciatic nerve in a reflex manner as well as over the area supplied by the posterior division of the nerve. It is not always possible to distinguish this pain from that caused by irritation or compression of the lumbar or sacral nerve roots, although these difficulties do not make it impossible to arrive at an accurate diagnosis. However, it does mean that a fairly accurate knowledge of the detailed anatomy of the lower back, including the distribution of all nerves and the function of all muscles, is required. Other information, such as the evolutionary changes in man as it applies to the lower back will be most valuable.

It is difficult to determine whether or not the many luxuries of modern living have been a factor in the increased incidence of low back injuries. There can be no question but that the loss of muscle tone under such conditions has made the back less resistant to injuries. Sir Arthur Keith[1] has pointed out that it is not correct to say that our spines are not perfectly adapted to the upright posture, but that it is more accurate to say that human spines were not evolved to withstand the monotonous and trying postures entailed by modern education and by many modern industries.

These and other predisposing factors make the lower back one of the areas most susceptible to injury. The change from quadruped posture to the erect has placed the lower back at the base of a long lever. The evolutionary changes which accompanied this change have further weakened the area because they have not

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been complete. The lumbar spine has shortened and the pelvic base has widened, with thickening and shortening of the muscle structure, but when this process is not complete one finds many variations or anomalies. As Willis[6] has indicated, these anomalies have been the result of an attempt to include the last lumbar vertebra in the sacrum. These include complete or partial sacralization of the transverse processes, narrow intervertebral disc (nontraumatic), asymmetry of the planes of the articular facets and failure of fusion of the posterior elements of the bony structures, such as separation of the neural arch and spina bifida. Each of these variations makes the lower back more vulnerable to injury.

Our experience has shown that asymmetry of the articular facets is the most frequent congenital variation found associated with low back disorders. They were found in 55 per cent of a series of 91 lumbosacral fusions reported by the author. The initiating or exciting causes of most low back injuries will result more from some faulty mechanics in lifting or over lifting with a resultant strain of the soft tissues of the back rather than from a fall. Most low back injuries occur just as a heavy object is being raised from a low position or when it is being lowered to such a spot. There is much increased muscle pull required to initiate and to terminate the act of moving a heavy object.

Evidence of some previous low back injury, such as narrowing of the intervertebral disc or degenerative arthritis in the intervertebral and apophyseal joints, presents one of the most difficult diagnostic problems. Certainly they make the low back much more prone to injury and at times they may be involved in total disability when a very mild injury, or no injury at all, has occurred. The act of bending or twisting in the absence of any strain may be accompanied by a sudden catch and severe pain in such cases. It is difficult for the physician to determine if such an incident is the result of an injury when it occurs while the individual is at work. The true diagnosis becomes more confusing when there is the added element of compensation. If a dilemma is to be avoided in these cases, it will be necessary to formulate a logical approach to the problem as it is presented in each case. We have attempted to do this by setting up the following criteria: Some accident must have occurred, or some unforeseen incident resembling an accident, which would produce an injury. This must be accompanied by at least momentary interruption of work. An examination of such a back should reveal some objective symptoms of demonstrable functional disturbance.

CLASSIFICATIONS

The structures in the lower back that are subject to injury have been mentioned in the discussion of complexity of the problem. These may be classified into two main groups:

A. Muscle and ligament strains
   1. Sacrospinalis muscles
   2. Gluteal muscles
   3. Lumbosacral and sacroiliac ligaments
   4. Interspinous ligaments
   5. Muscle aponeuroses or tendons

B. Joint injuries
   1. Strain of vertebral and intervertebral joint ligaments
   2. Strain of ligaments of apophyseal joints
   3. Traumatic arthritis
      a. Narrow intervertebral disc
      b. Changes in apophyseal joints

Any of these injuries may manifest themselves by an immediate disabling pain. However, most strains of the soft tissues will be followed by pain of short duration rarely completely disabling the patient at the time, but will recur as soon as stasis or local congestion occurs. This pain usually is severe enough to cause the individual to stop work, and it takes place a few hours after injury. On the other hand, disabling symptoms may not occur for varying intervals following the injury. This often is true of injuries that later result in narrowing of the intervertebral discs, weakening of the annulus fibrosis, or traumatic arthritis in the apophyseal joints. These cases may sustain a herniated nucleus pulposus or impingement of the articular facets by some unusual motion or mild strain. Rarely does a single acute injury produce a herniated nucleus pulposus at the time. Most often, an injury, even though mild, that has been superimposed on a pre-existing weak back, will recover very slowly.

DIFFERENTIAL DIAGNOSIS

The presence of some congenital anomaly or degenerative arthrosis of the lower spine does not indicate that either is the cause of disability following an injury. However, the existence of one or more such lesions may make the back more susceptible to injury. An accurate history and the physical findings form the real basis for making a correct diagnosis[3]. Data obtained from the laboratory may or may not be of any value. One has to be extremely careful to interpret such findings, particularly the roentgenogram, in terms of the clinical findings. Those of you involved in industrial medicine are often confronted with the problem of deciding whether some pre-existing lesion is the cause of the disability or whether some recent compensable injury is present.

Acute myofascial or ligament strains usually will have localized back pain and trigger point tenderness. Often the motion will be moderately limited with some muscle spasm, but this motion will not be very painful. At times the pain will be referred over either buttock and down the course of the sciatic nerve; however, signs of nerve root compression will be absent. The relief of pain following the injection of novocain into the trigger area will confirm the diagnosis. The presence of some congenital or structural weakness often will be an important factor in the prognosis.

As the lesions become subacute, chronic muscle contractures will develop and the tenderness will become more diffuse. This will be accompanied by a gradually progressing muscle decompensation and increasing lordosis with a resultant chronic strain on the articular synovia or capsule. The latter condition manifests itself by soreness and stiffness in the morning with little pain at the time, but the pain will increase with activity and is most severe for one to two hours after

retiring. The latter pain is a rest pain that is produced by stretching of the shortened ligaments and muscles that accompanies the lordosis.

Acute joint injuries will be accompanied by much pain on attempted motion. The muscle spasm will be marked, all muscles being very tense so as to prevent motion. When this lesion becomes chronic it will present the same features and findings seen in chronic myofascial strains.

Posterior herniation of the nucleus pulposus may occur following an acute severe injury, but oftener it will follow some very minor strain or merely some twisting motion. The immediate complaint is pain in the lower back which at times is followed almost at once by sciatic pain, while at other times the radiated pain may not develop for a few days. The pain may go into the calf of the leg as far as the heel and to the sole of the foot and the big toe or it may be referred along the outer side of the foot to the small toes. Usually this pain distribution will correspond to the nerve segment or segments involved; however, at times it is difficult to distinguish it from the referred pain in low back sprains. Therefore, the diagnosis cannot be made on the distribution of the sciatic pain. There are certain findings that a careful examination will elicit and, if none of these are present, the author believes that a diagnosis of a herniated nucleus pulposus cannot be made. These may be ennumerated as follows: Atrophy of the calf muscles, some hypesthesia of the nerve segment that is involved, weakness of the intrinsic muscles of the foot, and decreased or absent ankle jerk or posterior tibial reflex. Our prerequisite has been that one or more of these findings must be present to make a positive diagnosis. The myelogram may be of some help, but to make a diagnosis on the presence of a defect alone will result in many errors. We do not use the myelogram as a diagnostic measure, but rather to localize the lesion and determine if multiple lesions exist.

TREATMENT

The treatment of low back injuries, to be effective, always presupposes a correct diagnosis, particularly as to the tissues involved. However, since this is not possible in a great number of cases, one has to proceed cautiously. All conservative means available should be employed, resorting to surgery only after these have failed. It is our opinion that the only indication for immediate surgery is a herniated nucleus pulposus with marked cord or nerve root compression. Conservative procedures, if adequate, and if continued for a sufficient length of time, will give relief in 95 to 98 per cent of the patients. The presence of an unstable back from a spondylolysis, spondylolisthesis, asymmetrical facets, transitional vertebrae, narrow intervertebral disc, or degenerative arthritis of the articular facets in itself is not an indication for operation. Most individuals with such lesions will go through life with little disability. Even many persons with an injury superimposed upon any of these pre-existing conditions will obtain complete relief from conservative measures.

Strains involving any or all of the structures in the lower back should be protected during the acute stages. Novocain may be injected into the trigger areas to relieve pain and muscle spasm. The back then should be taped or placed

in a well-fitted corset if the strain is mild, but the back should be placed at complete rest in bed if the strain has been severe. The treatment is directed toward relief of pain and muscle spasm during the acute stage. Moderate flexion of the knees and hips in a firm Ford bed allows correction of the lordosis and tends to relieve the strain. Curare in oil will be of value, but its use requires very close supervision by hospital personnel. Only moist or radiant heat should be used at this time because induction heat will very often aggravate the symptoms. The patient who has had severe pain always is kept at complete rest for several days after all symptoms have subsided. Massage and graduated exercises are begun at this time. Failure to follow up these patients with exercises to loosen up and restore the muscle tone often leads to chronic low back pain. Those patients in whom the injury has been superimposed on a structurally weak back are fitted with a rigid back support. This is worn during the period of rehabilitation and discarded when the back has recovered.

Patients with a suspected or definite herniated nucleus pulposus in which the nerve root irritation or compression is mild are treated by the same methods. They usually are kept at complete bed rest for a longer period of time and the back is protected from any strain until all signs and symptoms have disappeared. Most of these will respond well and will have no further recurrence. Others will obtain relief with rest, but will have recurrences with return to normal activity. These patients respond well to surgery. It has been our experience that the patient, other than the one with a large acute disc, who does not obtain appreciable relief from a regime of complete bed rest and physical therapy, is not likely to respond well to surgery. The poorest results from surgery have been obtained in this group. It is now our policy to advise against surgery in such patients. Even in those patients in whom we think surgery is indicated the final decision always is left entirely up to the patient. Our results have been excellent in those patients in whom compensation is not involved, but not very good when compensation is implicated. These results correspond to those reported by Voris[4] who obtained 83 per cent good results in noncompensation patients and only 37 per cent good results in the compensation patients.

There has been much controversy as to the type of surgery indicated in low back pain. Some authors have believed that practically all low back disability arises from lesions of the intervertebral discs and that removal of the nucleus pulposus is all that is required. Others believe that the disc should be removed only when nerve root symptoms are present, while still others are of the opinion that every patient should have also a low back fusion. It has been our experience that those patients who have chronic low back pain associated with a herniated nucleus pulposus and who get relief from conservative measures but which recurs to a disabling degree with activity should have a removal of the nucleus pulposus and a lumbosacral fusion.[2] Removal of the disc alone in these patients may relieve the root pain but will not relieve the back pain.

SUMMARY AND CONCLUSIONS

The complex anatomy, inaccessibility of many structures in the lower back, and the similarity of symptoms found in injuries to the various structures make

it difficult to evaluate injuries to this part of the body. The ability to evaluate these injuries presupposes a fairly complete knowledge of its anatomy, and the variations that occur therein as well as the mechanics involved in low back function.

The diagnosis of an injury to the low back must be based on evidence that some accident, or incident that would produce an injury, has taken place. In order to make a correct diagnosis, it is necessary to have an accurate, detailed account of this accident. The pathology in a back that is disabled from a strain is quite different from that found following some simple or bending motion that involves no strain. This history is most important in interpreting the physical and laboratory findings.

The type and severity of the injury to the lower back as well as the presence or absence of some pre-existing lesion will determine the treatment. However, every injured back should be protected from further strain until recovery has reached its maximum. All conservative measures should be exhausted before resorting to surgery. Those patients who obtain marked improvement under conservative management, but who have a recurrence of symptoms upon returning to active life, will respond well to surgery. Chronic low back pain with or without sciatica, which does not get temporary relief from adequate conservative measures, will get a poor result from surgery in most instances. It should be noted that these patients quite often give only a vague history of injury. Finally the type of surgery indicated should be determined by the pathology that is present.

REFERENCES

1. KEITH, SIR ARTHUR: The Injured Back and Its Treatment, Edited by J. D. Ellis, Springfield, Illinois, 1940, p. 16.
2. OVERTON, L. M.: Arthrodesis of Lumbosacral Spine, Clinical Orthopaedics (in press).
3. STEINDLER, A.: Differential diagnosis of pain low in back, J. A. M. A. *110:* 106 (Jan. 8) 1938.
4. VORIS, H. C.: End results of disc operation in industry, J. Internat. Coll. Surgeons *2:* 198 (Feb.) 1954.
5. WATSON-JONES, SIR REGINALD: Fractures and Joint Injuries, 3rd. ed. Vol. I, Baltimore, The Williams and Wilkins Company, 1946.
6. WILLIS, T. A.: Analysis of vertebral anomalies, Am. J. Surg. *6:* 163 (Feb.) 1929.

RECURRENT OR PERSISTENT PEPTIC ULCERATION FOLLOWING SECONDARY OPERATIONS FOR PEPTIC ULCER

AN ANALYSIS OF ELEVEN CASES

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Although the initial definitive surgical treatment of the ulcer diathesis fortunately is effective in the majority of instances, in a small percentage of cases persistent or recurrent peptic ulceration signally indicates failure of the primary surgical treatment. Most patients in whom the primary surgical treatment has proved ineffective obtain satisfactory control of the ulcer diathesis following an appropriate secondary surgical procedure. Yet, there still may remain a small group of patients who continue to have persistent or recurrent peptic ulceration even after a secondary operation. Some patients in this latter group may require several secondary surgical procedures before control of the peptic ulceration is obtained and, unfortunately, in a few patients control of the ulcer diathesis is not satisfactorily obtained even by repeated secondary surgical procedures.

In a group of 90 patients with gastrojejunal ulceration treated at the University of Illinois Research and Educational Hospital and the Hines Veterans Administration Hospital during the period 1946–1954 there were 11 patients who had persistent or recurrent ulceration following secondary operations. An analysis of these 11 cases is the subject of this report.

CASE REPORTS

Case 1. R. S., a white male born in 1909 first noticed the onset of ulcer symptoms in 1944. In 1947 an emergency operation consisting of *ligation of gastric vessels and pyloroplasty* was done for upper gastrointestinal hemorrhage. On Aug. 1, 1947 a *subdiaphragmatic vagotomy* was done. Postoperative tests indicated an incomplete vagotomy. The patient had recurrence of pain within four months after this operation. A cholecystectomy was done on Feb. 18, 1948 for a poorly functioning gallbladder, without relief of abdominal symptoms. On Nov. 30, 1948 a ruptured appendix was removed at Hines Veterans Administration Hospital, and during this admission a duodenal ulcer was demonstrated by roentgenologic examination. In 1949 a *partial gastric resection* was done at the Marine Hospital in Chicago because of persistent ulcer symptoms. On June 27, 1950 the patient was hospitalized at Hines Veterans Administration Hospital and treated conservatively for small bowel obstruction. During this hospital admission he had six to seven loose stools per day. Two weeks after discharge from the hospital he returned with symptoms of severe diarrhea, nausea, vomiting, and severe right lower quadrant pain. Diagnostic studies showed a gastrojejunocolic fistula. In July 1950 a *resection of the gastrojejunocolic fistula*, a *higher gastric resection*,

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and a *colostomy* were done. The colostomy later was closed on Nov. 17, 1950. He has been asymptomatic since the last operation. The date of last follow-up was July 1954.

Comment. This patient was subjected to four operations because of his ulcer diathesis. The first operation i.e., ligation of gastric vessels and pyloroplasty apparently was utilized to control an acute bleeding episode and is not generally accepted as an effective method of treating the ulcer diathesis. A subdiaphragmatic vagotomy done the same year was found by postoperative tests to be incomplete and likewise was ineffective in relieving ulcer symptoms. A partial gastric resection approximately two years later was limited in extent and failed to provide noticeable improvement. Finally in July 1950, after the development of a gastrojejunocolic fistula a higher gastric resection and resection of the fistula were done. The patient has been to date asymptomatic for four years since the last operation. The failures of vagotomy and gastric resection in this patient properly may be ascribed to the fact that these operations were initially inadequately done. An adequate (75 per cent) gastric resection at the time of the primary surgical procedure might well have obviated the need for further surgical treatment of the ulcer diathesis.

Case 2. M. B., a white female born in 1900 first noticed the onset of ulcer symptoms in 1933. Roentgenograms in 1938 demonstrated a duodenal ulcer. Because of persistent ulcer pain a *gastroenterostomy* and closure of the duodenum were done in 1938. She then had relief of symptoms until 1950 when she noted gaseous distention, vomiting, abdominal pain, and intractable diarrhea. A gastrojejunocolic fistula was diagnosed and the *fistula* was *excised* in 1950. In 1952 she developed obstruction at the gastrojejunal stoma and the *segment of jejunum* was *resected* and a new *gastroenterostomy* done. Postoperatively she did fairly well except for frequent episodes of emesis and abdominal pain which were relieved by food and alkali. In January of 1953 she developed sudden severe right upper quadrant abdominal pain radiating into the left upper and the right lower quadrants of the abdomen. An exploratory celiotomy was done and a perforated jejunal ulcer was found at the site of the gastrojejunostomy. The *perforation* was *closed*. Following this procedure she had symptoms of obstruction with frequent vomiting. On April 10, she had abdominal pain, emesis, and tarry stools. Copious vomiting and increased bowel sounds were present although there was no cramping pain. She was treated by continuous gastric suction. On April 18, she was operated upon. At operation the anastomosis between the stomach and the jejunum was found to be densely scarred. The *stoma* was *revised*. Because of dense adhesions between the stomach and the chest wall and liver a vagotomy was not done at this time. On May 1, a *transthoracic vagotomy* was done. She was discharged from the hospital on May 16. Insulin tests postoperatively indicated a complete vagotomy. Roentgenologic examination on Dec. 4, 1953 showed a normally functioning gastroenterostomy with no evidence of obstruction or marginal ulcer. This patient was examined last in July 1954. She has been asymptomatic since the last operation.

Comment. Five surgical procedures were done upon this patient before a defini-

tive attack was made upon the ulcer diathesis. Following a complete transthoracic vagotomy she has been asymptomatic to date for over a year.

Case 3. G. S., a white male born in 1895 first noticed the onset of ulcer symptoms in 1920. In 1926 a *posterior gastroenterostomy*, cholecystectomy and appendectomy were done because of persistent abdominal pain. Almost immediately following operation he complained of nausea, vomiting, and "heaviness" in the stomach. These symptoms persisted until 1934 when he had his first episode of massive upper gastrointestinal hemorrhage. He had another episode of massive upper gastrointestinal hemorrhage in July 1935 at which time he was admitted to the Hines Veterans Administration Hospital and was treated medically. In August 1936 he again was admitted to the Hines Veterans Administration because of stomal ulcer manifested by pain. In April 1941 he had another hemorrhage and was treated medically at the Hines Veterans Administration Hospital. In August 1943 he had another upper gastrointestinal hemorrhage which was treated medically. In 1945 a *partial gastric resection*, estimated to be approximately a 35 per cent resection, was done at a private hospital. He was well until December 1949 at which time he had another upper gastrointestinal hemorrhage and again was treated medically. In May of 1950 he had another bleeding episode which required hospitalization. In December 1950 he refused operation for treatment of another upper gastrointestinal hemorrhage and responded to medical therapy. In June of 1951 he was readmitted to the Hines Veterans Administration Hospital because of upper gastrointestinal hemorrhage and a *transthoracic vagotomy* was done. Postoperative studies indicated that the vagotomy was incomplete, and in September 1951 he was readmitted to Hines Veterans Administration Hospital because of upper gastrointestinal hemorrhage and obstruction. On Feb. 14, 1952 a *subtotal gastric resection*, estimated to be approximately a 75 per cent resection, was done for a marginal ulcer and upper gastrointestinal hemorrhage. He has been asymptomatic since the last operation. The date of last follow-up was July 1954.

Comment. The initial surgical procedure in this patient was a gastroenterostomy which is known to be ineffective in a large percentage of cases in controlling the ulcer diathesis. The first two secondary procedures must be classified as inadequate, in the first instance because of the very limited extent of gastric resection, and in the second instance because of the incompleteness of the vagotomy. The patient has been asymptomatic for over two years following a 75 per cent gastric resection.

Case 4. D. T., a white male born in 1895 first noticed the onset of ulcer symptoms in 1931. On April 11, 1951 he was admitted to Hines Veterans Administration Hospital because of a massive upper gastrointestinal hemorrhage. He responded to medical management, but had repeated episodes of bleeding on July 17 and Sept. 5. On Oct. 16, 1951 a *vagotomy* and *gastroenterostomy* were done. At the time of operation a bleeding ulcer was noted at the junction of the first and second portions of the duodenum. Postoperative studies indicated an incomplete vagotomy, and the patient had almost immediate recurrence of pain postoperatively. On Jan. 1, 1953 he had another upper gastrointestinal hemorrhage which

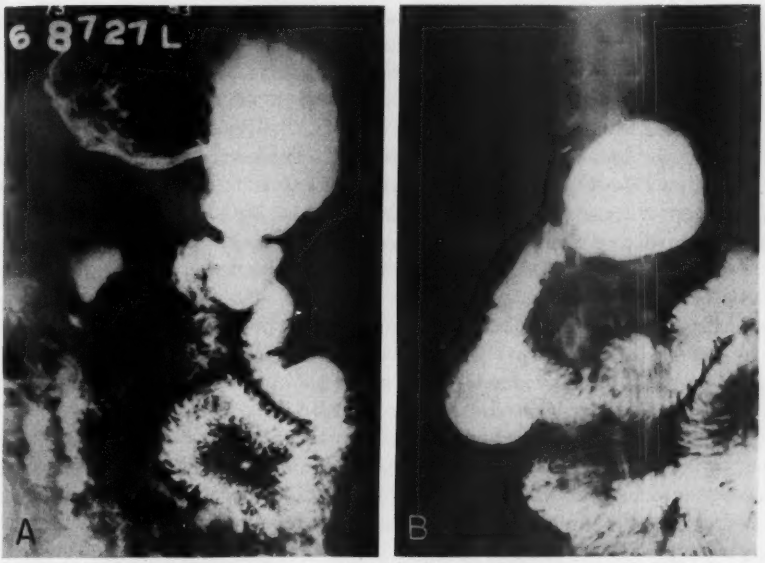

FIG. 1.—(Case 4.) A. Roentgenogram of residual stomach after a so-called gastric resection. B. Roentgenogram of residual stomach after gastric resection measured to be at least a 75 per cent resection.

was treated medically. On March 10, there was another upper gastrointestinal hemorrhage, and on March 25, a *partial gastric resection* was done below the gastroenterostomy. From inspection of roentgenograms of the stomach before and after this gastric resection it is estimated that not more than 25 per cent of the stomach was resected (fig. 1). At the time of operation a marginal ulcer was not found but an active duodenal ulcer was demonstrated. He again was readmitted to the hospital on August 10, with the complaint of left lower quadrant pain. Roentgenograms demonstrated the presence of a marginal ulcer. On Sept. 16, 1953 a *Billroth I type subtotal gastric resection* was done. The extent of the resection was measured to be 78 per cent. A marginal ulcer was demonstrated at operation. He has been asymptomatic since the last operation. The date of the last follow-up was July 1954.

Comment. The primary operation in this patient was a vagotomy and gastroenterostomy; however, the vagotomy was incomplete and accordingly the patient was in much the same status as if only a gastroenterostomy had been done. The first secondary procedure on this patient was an inadequate gastric resection of approximately 25 per cent of the stomach. Following a high gastric resection (approximately 78 per cent) the patient has been asymptomatic.

Case 5. P. M., a white male born in 1894 first noticed the onset of ulcer symptoms in 1918. In 1927 he had a *gastroenterostomy* for a penetrating duodenal ulcer. In 1928 he had an upper gastrointestinal hemorrhage. He had repeated upper gastrointestinal hemorrhages in June and September 1933, January 1936, and

May 1948. During the last two admissions he refused operation. In May 1948 a *subdiaphragmatic vagotomy* was done because of upper gastrointestinal hemorrhage. Postoperative tests indicated an incomplete vagotomy. The postoperative gastric acidity was 58 degrees of combined and 44 degrees of free hydrochloric acid. Despite the relatively high gastric acidity he remained well until June of 1949 when he had recurrent ulcer symptoms. There was, however, no bleeding at this time. He was placed on medical management. In May of 1951 he had another upper gastrointestinal hemorrhage and on Jan. 9, 1954 because of recurrent hemorrhage a *subtotal gastric resection*, estimated to be approximately a 75 per cent resection, was done. A marginal ulcer was demonstrated at operation and was excised with the specimen.

Comment. This patient had an incomplete vagotomy as the first secondary operation after the primary gastroenterostomy. He has been asymptomatic to date for approximately six months following a 75 per cent gastric resection.

Case 6. V. R., a white female born in 1889 first noticed the onset of ulcer pain at the age of 10 years at which time she had attacks of abdominal pain lasting two to three hours which sometimes were relieved by food. In 1929 she had a massive upper gastrointestinal hemorrhage and a *gastroenterostomy* was done in November 1929. In 1930 she had a massive upper gastrointestinal hemorrhage and a marginal ulcer was demonstrated. She had recurrent upper gastrointestinal hemorrhages at 3 to 18 months intervals until 1949 when on May 2, 1949 a *subdiaphragmatic vagotomy* was done. The postoperative studies indicated an incomplete vagotomy and on May 16, 1949 she had another gastrointestinal hemorrhage. In 1950 she had massive upper gastrointestinal hemorrhages, due to a marginal ulcer, on May 30, and on July 22. The marginal ulcer was demonstrated by roentgenograms. On Aug. 8, 1950 a *subtotal gastric resection*, estimated by the surgeon to be approximately a 75 per cent resection, was done. She has been asymptomatic since the last operation. The date of last follow-up was July 1954.

Comment. This patient also had an incomplete vagotomy as the first secondary operation after recurrence of upper gastrointestinal hemorrhage following the primary procedure of gastroenterostomy. She has been asymptomatic to date for approximately four years following a 75 per cent gastric resection.

Case 7. E. H., a white male born in 1897 first noticed the onset of ulcer symptoms in 1922. He was essentially symptom free from 1928 to 1940 during which period he took various patent medicines. During 1940 he was subjected to severe emotional distress and the ulcer symptoms returned. Abdominal pain then occurred off and on until July 1947 when roentgenograms demonstrated the presence of cholelithiasis and duodenal ulcer. In June 1948 a cholecystectomy was done. In July 1948 the abdominal pain recurred and was accompanied by frequent vomiting. This pain was not relieved by antacids or by food. In October 1948 a *partial gastric resection* was done because of partial pyloric obstruction. He had two episodes of upper gastrointestinal hemorrhage during February 1949. He was treated medically, and a marginal ulcer was demonstrated by roentgenograms on April 6, 1950. On May 5, an exploratory celiotomy was done at the University of Illinois Research and Educational Hospital and a marginal ulcer was identified perforating into the liver. This *perforation* was *closed*. The previous

gastric resection had not been very extensive as approximately ¾ of the stomach remained. On May 12, he had a wound dehiscence which required *operative closure*. Postoperative roentgenograms showed a tremendous dilatation of the proximal segment of the duodenum and jejunum proximal to the gastrojejunostomy, and on June 15, a *subtotal gastric resection*, estimated by the surgeon to be an 80 per cent resection, was done. Not more than 5 cm. of stomach was left. The gastrojejunal ulcer removed at operation measured approximately 4 cm. in diameter. On July 2, he complained of persistent pain in the epigastric region. He had several episodes of massive gastrointestinal hemorrhage requiring multiple transfusions. No ulcer was demonstrated by roentgenogram. Measurement of the surface area of the barium outlined residual stomach indicated that the patient had had a 75 per cent resection. He continued to complain of epigastric pain and vomiting. Gastroscopy showed a marginal ulcer. The gastric acidity was 21 degrees of combined and 13 degrees of free hydrochloric acid. A *transthoracic vagotomy* was done on Oct. 19, 1950. He has been asymptomatic since the last operation. The date of the last follow-up was July 1954.

Comment. This patient had several upper gastrointestinal hemorrhages and a perforation of a marginal ulcer after the limited gastric resection. Following a secondary subtotal gastric resection, estimated and confirmed by measurement of the surface area of the barium outlined stomach to be at least a 75 per cent resection, there was a recurrence of ulcer symptoms. It is interesting to note that the patient's gastric secretions were not rendered achlorhydric by this operation. Following the transthoracic vagotomy he has been asymptomatic to date for almost four years.

Case 8. A. W., a white male born in 1889 first noticed the onset of ulcer symptoms in 1927. In 1934, because of persistent ulcer symptoms, a *gastroenterostomy* was done. He had almost immediate recurrence of ulcer symptoms. On Dec. 6, 1947 a *subtotal gastric resection* was done because of duodenal and marginal ulcerations. The resection was estimated by the surgeon to be approximately 65 per cent of the stomach. In September 1949 he had an upper gastrointestinal hemorrhage. Diagnostic studies, including roentgenogram, esophagoscopy, and gastroscopy failed to demonstrate the cause of bleeding. He was placed on medical management. He was admitted to the hospital on July 9, 1950 with upper gastrointestinal hemorrhage and abdominal pain. The diagnosis of marginal ulcer was made at this time.

Comment. This patient had a recurrence of ulcer symptoms after a primary gastroenterostomy and a secondary subtotal gastric resection estimated by the surgeon to be a 65 per cent resection. The patient refused further surgery. One can only conjecture as to whether a higher (75 per cent) resection or complete vagotomy would have controlled the ulcer diathesis in this patient.

Case 9. E. C. C., a white male born in 1894 first noticed the onset of ulcer symptoms in 1917. In 1921 he had a perforation of a duodenal ulcer which required *closure*. In 1925 a *partial gastric resection* (*pylorectomy*) was done. Fourteen months later he had a massive upper gastrointestinal hemorrhage and since that time upper gastrointestinal hemorrhages have occurred at least once per year. He was admitted to Hines Veterans Administration Hospital on April 4,

1939 because of a massive upper gastrointestinal hemorrhage. He was given medical treatment and operation was recommended but refused by the patient. He was readmitted to Hines Veterans Administration Hospital on April 16, 1942 with another episode of upper gastrointestinal hemorrhage and again was controlled by medical management. On Nov. 26, 1946 he again was admitted to Hines Veterans Administration Hospital because of massive upper gastrointestinal hemorrhage. An *exploratory celiotomy* was done at this time. An ulcer was not visualized because of adhesions and inflammatory reaction, and the abdomen was closed without any definitive surgery. The patient made an uneventful postoperative recovery, and on July 14, 1947 a *transthoracic vagotomy* was done. Postoperative studies indicated an incomplete vagotomy. Because of another massive upper gastrointestinal hemorrhage a *higher gastric resection* was done on Oct. 27, 1947. However, the operative report indicated that only 4 cm. of the stomach was resected. Ulcer pain occurred immediately after operation and six weeks later he had a minor upper gastrointestinal hemorrhage. On April 29, 1949 he was admitted to the hospital for massive upper gastrointestinal hemorrhage and was treated medically. On May 3, Nov. 11, 1949 and July 31, 1950 he was readmitted to the hospital for upper gastrointestinal hemorrhages. He had been on strict medical management since early in 1948. Despite rigid medical management, however, he continued to have ulcer pain and recurrent upper gastrointestinal hemorrhages. On follow-up by letter in January 1954 he was found to be living in Arizona and to be following rigid medical management, but still to be having typical ulcer pain and to have had several hemorrhages since his last admission to the Hines Hospital in 1950. The patient is reluctant to submit to further surgery in view of the fact that he has already had two gastric resections and a transthoracic vagotomy without cessation of either ulcer pain or gastrointestinal hemorrhage.

Comment. Although this patient has had two gastric resections and a transthoracic vagotomy without relief of ulcer symptoms it must be noted that both gastric resections were limited in extent and the vagotomy was demonstrated by postoperative studies to be incomplete. The patient's lack of confidence in further surgery is easily understandable.

Case 10. E. C., a white male born in 1893 first noticed the onset of ulcer symptoms in 1916. In 1933 he had a perforation of a duodenal ulcer. The perforation was closed. In 1945 a *gastroenterostomy* was done, and he had an immediate return of ulcer symptoms following this procedure. In May 1946 he had an upper gastrointestinal hemorrhage which was treated medically at the Hines Veterans Administration Hospital. In October 1946 a vagotomy was advised and refused by the patient. In 1947 *resection of a stomal ulcer* and a *gastroenterostomy* were done. He had immediate recurrence of ulcer symptoms consisting of pain and repeated upper gastrointestinal hemorrhages. On June 17, 1951 an active jejunal ulcer was demonstrated by roentgenogram and was treated medically. On July 23, Oct. 26, 1951, and on September 3, and May 19, 1952 he was re-hospitalized for recurrent upper gastrointestinal hemorrhages. On May 27, 1952 a *subtotal gastric resection* was done. He developed anuria and jaundice and died postoperatively.

Comment. This patient had two gastroenterostomies done before a definitive operation was undertaken to control the ulcer diathesis. Subtotal gastric resection was done after the patient had had repeated upper gastrointestinal hemorrhages over a period of six years. He died following operation. This fatality might have been avoided had a definitive procedure been done at the time of the primary operation.

Case 11. D. B., a white male born in 1910 had had ulcer symptoms for a number of years before 1946. On July 19, 1946 a *gastric resection,* estimated by the surgeon to be a 60 per cent resection, was done. On August 8, he had a perforation of a stomal ulcer which required *surgical closure.* Following this procedure he had a massive upper gastrointestinal hemorrhage with perforation of two jejunal ulcers. *Resection of the involved jejunum* and an *anterior gastrojejunostomy* were done on August 11. On September 15, he complained of epigastric pain and a marginal ulcer was demonstrated by roentgenogram. He was treated medically. On Dec. 9, 1946 because of persistent pain due to a large lesser curvature ulcer a *subdiaphragmatic vagotomy* was done. No record as to the completeness of the vagotomy was available. On March 24, 1948 he complained of epigastric pain and had an upper gastrointestinal hemorrhage. On March 27, a *huge greater curvature ulcer* extending into the jejunum *was excised.* On March 31, a gastrojejunocutaneous fistula developed. He was admitted to Hines Veterans Administration Hospital on April 22. On July 21, *a resection of the fistulous tract and revision of the gastroenterostomy* were done. He developed a postoperative intestinal obstruction and on Sept. 27, 1948 a *celiotomy and jejunostomy* were done. Postoperatively the patient had intermittent upper gastrointestinal hemorrhages and died on Jan. 14, 1949 of hemorrhage and malnutrition. At autopsy a large gastric ulcer was found to be eroding into the splenic vein and artery and also to be communicating with the transverse colon.

Comment. This patient had six surgical procedures done following an initial gastric resection estimated by the surgeon to be a 60 per cent resection. However, only one of these six secondary procedures (subdiaphragmatic vagotomy) might be expected to favorably influence the ulcer diathesis, and there is no record as to whether or not the vagotomy was complete. This patient had two stomal ulcer perforations following the primary operation. Definitive attack on the ulcer diathesis (increasing the extent of gastric resection to a minimum of 75 to 80 per cent and possibly to a near total resection) well might have yielded a more favorable outcome than the simple excision of the marginal ulcer which was done in March 1948. The postoperative complications following resection of the stomal ulcer in March 1948 apparently prevented a later definitive surgical attack on the ulcer diathesis, and the patient died from an upper gastrointestinal hemorrhage. Again one might conjecture that the long series of secondary operations and eventual fatal outcome might have been obviated had a more extensive (75 per cent) gastric resection been done initially.

DISCUSSION

In this series of 11 patients a total of 38 operations, presumably intended to control the ulcer diathesis, were done. The number of such operations per patient

varied from a minimum of two to a maximum of five in 2 patients. In addition four operations were done for closure of ulcer perforations. Review of the case reports, however, indicates that only 24 of the 38 operations were procedures generally accepted to be effective in controlling the ulcer diathesis; i.e., gastric resection or vagotomy.

Vagotomy was done nine times in this series; however, the vagotomy was found postoperatively by the insulin test to be complete in only 2 patients. These 2 patients had relief of their ulcer symptoms. Gastric resection was done 15 times; but in only 7 instances was 75 per cent or more of the stomach resected. In 5 of these 7 patients control of the ulcer diathesis was obtained. One patient died postoperatively after an adequate gastric resection, and 1 patient (case 7) had recurrence of ulcer symptoms after a resection estimated by the surgeon to be an 80 per cent resection. The extent of the gastric resection, in this case, was confirmed by measurement of the surface area of the barium outlined residual stomach to be at least 75 per cent. This patient had relief of symptoms after a complete transthoracic vagotomy. In 8 of the gastric resections less than 75 per cent of the stomach was removed. It is interesting to note that in none of these 8 resections did the surgeon believe that a 75 per cent gastric resection had been done. The surgeon's estimate of the extent of resection in these 8 cases varied from pylorectomy to a 65 per cent resection.

Thus, of the 11 patients in this series, 2 finally had relief of symptoms following complete vagotomy and 5 obtained control of the ulcer diathesis following an adequate (75 per cent) subtotal gastric resection. Two of the remaining 4 patients died; 1 patient (case 10) died of anuria following an adequate subtotal gastric resection after having had two previous inadequate surgical attacks upon the ulcer diathesis; another patient (case 11) died after having had four secondary surgical procedures following an initial gastric resection estimated by the surgeon to be a 60 per cent resection. Two patients continue to have ulcer symptoms and have refused further surgery. One of these patients (case 9) has had two gastric resections, both limited in extent, and an incomplete vagotomy. This patient's lack of confidence in surgeons and further surgery is easily understandable.

Wangensteen and his co-workers[1, 4, 5] have demonstrated that gastrectomy is effective in preventing histamine-provoked ulceration in experimental animals only if at least 75 per cent of the stomach is resected. Their clinical incidence of 11 recurrences (1.5 per cent) in 732 patients subjected to a 75 per cent resection clearly substantiates their experimental data.[7] We recently have re-emphasized the necessity for more accurate estimation of the extent of gastric resection to ensure an adequate gastric resection and minimize the incidence of recurrent or persistent ulceration.[2] A method which correlates the amount of stomach resected and the amount of stomach remaining, such as the polar planimeter method of Harkins and Moore, is recommended.[6] A simpler method of comparing the lengths of the lesser and greater curvatures of the original and residual stomach also is useful but probably not as accurate as the method of Harkins and Moore. Although estimating the extent of the resection by weighing the resected specimen might

be expected to be subject to appreciable error in the presence of edema and hypertrophy, the method has been used by Wangensteen and his co-workers[7] with admirable results.

Just as precautions must be utilized to ensure a 75 per cent gastric resection, if that be the procedure of choice, also care must be taken to increase the incidence of completeness of vagotomy if that procedure is employed. The incidence of complete vagotomy will be increased if this procedure is done transthoracically. In as much as revision of the gastroenterostomy stoma frequently is necessary at the time of a secondary procedure, operation via the abdominal route may be required. If such is the case, the vagus nerves should be divided as high on the esophagus as possible after the lower portion of the esophagus has been mobilized from the diaphragmatic crura. Unfortunately, because of anatomic variations, the vagotomy occasionally will not be complete despite the careful performance of this operation.

With a 75 per cent gastric resection, primary or secondary control of the ulcer diathesis will be obtained in the majority of instances. The few patients who develop recurrent or persistent ulcer symptoms following adequate gastric resection will respond to complete vagotomy in most cases. Those few patients who do not respond to the combination of these two operative procedures may require near total gastric resection or, perhaps instead, extensive medical therapy including Banthine as suggested by Glenn.[3]

CONCLUSIONS

In a group of 90 patients with gastrojejunal ulceration treated at the University of Illinois Research and Educational Hospital and the Hines Veterans Administration Hospital during the period 1946 to 1954 there were 11 patients who had persistent or recurrent ulcerations following secondary operations.

In the series of 11 patients 38 operations, presumably intended to control the ulcer diathesis, were done. Only 24 of the 38 operations were procedures generally accepted, at the present time, to be effective in controlling the ulcer diathesis.

Vagotomy was done nine times in this series; however, the vagotomy was found postoperatively by the insulin test to be complete in only 2 patients. These 2 patients had relief of their ulcer symptoms. Gastric resection was done 15 times; in only 7 instances, however, was 75 per cent or more of the stomach resected. In 5 of these patients control of the ulcer diathesis was obtained. One patient died postoperatively and the other patient required a vagotomy before cessation of ulcer symptoms was obtained.

Persistent or recurrent ulceration following secondary as well as primary operations for the ulcer diathesis is in most instances due to inadequacy of the extent of gastric resection or to incompleteness of vagotomy. If ulceration occurs after adequate (75 per cent) gastric resection, complete vagotomy will control the ulcer symptoms in the great majority of instances.

REFERENCES

1. Baronofsky, I., Lannin, B. G., Sanchez-Palomera, E., and Wangensteen, O. H.: Billroth I gastric resection; extent necessary to protect against histamine-provoked ulcer, Proc. Soc. Exper. Biol. & Med. *59:* 229 (June) 1945.
2. Everson, T. C.: More accurate estimation of extent of gastric resection, Editorial, Ann. Surg. *140:* 260 (Aug.) 1954.
3. Glenn, F.: Second operations for peptic ulcer, Ann. Surg. *132:* 702 (Oct.) 1950.
4. Lannin, B. G.: Experimental evaluation of a satisfactory operation for ulcer, Surgery *17:* 712 (May) 1945.
5. Lannin, B. G., Hay, L. J., Judd, E. S., and Wangensteen, O. H.: Evaluation of satisfactory operation for ulcer, Proc. Soc. Exper. Biol. & Med. *56:* 231 (June) 1944.
6. Moore, H. G., Jr., and Harkins, H N.: Critical evaluation of Billroth I gastric resection, Surgery *32:* 408 (Aug.) 1952.
7. Rauch, R. F.: Evaluation of gastric resection for peptic ulcer, Surgery *32:* 638 (Oct.) 1952.

SURGICAL COMPLICATIONS RESULTING FROM ACTH AND CORTISONE MEDICATION

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With the increasing use of ACTH, cortisone and other adrenal steroids, it is well to bear in mind that their use may precipitate complications requiring surgical intervention. Also, in addition to their many beneficial effects in surgical patients,[1] their use prior to surgery, if unknown to the surgeon, may cause acute adrenal cortical failure and death in the postoperative period.[3, 7, 10, 14] The ease with which the oral preparations of these drugs are available and administered makes it advisable to question every surgical patient about their use before operation.

The adrenocorticotrophic hormone (ACTH) is produced by the anterior pituitary gland probably by stimulus from the hypothalamic region. ACTH in turn stimulates the adrenal cortex to secrete the adrenal steroid compounds. There are thought to be some 28 of these steroids, only 6 of which have biologic activity. Normally, the adrenal cortex of the human being secretes a large preponderance of a single steroid—17 hydroxy-corticosterone (Compound F). The biologic activity of 17 hydroxy-corticosterone for all practical purposes is quite similar to cortisone; thus, one might think of ACTH as stimulating the adrenal cortex to secrete cortisone.

The physiologic action of cortisone and the other adrenal steroids has been reviewed thoroughly by many authors. Probably the most complete recent publication is the series of articles in the New England Journal of Medicine by Thorn and associates early in 1953.[13]

In the past two years we have seen a number of surgical complications resulting from the use of ACTH and cortisone.

PEPTIC ULCER

Cortisone has been shown both in normal patients and patients with peptic ulcer to cause an increase in the basal and nocturnal hydrochloric acid secretion, associated with a marked rise in pepsin and pepsinogen. The urinary excretion of uropepsin in normal pateints often is raised to the high levels encountered in patients with peptic ulceration.[13] This brings two clinical facts to mind—the marked increase in appetite noted in patients on ACTH or cortisone and the deleterious effects that were found in the treatment of gastric ulcers with ACTH in a few human subjects.

The development of gastric or duodenal ulcers or the aggravation of existing ulcers in patients under ACTH or cortisone therapy has been reported by many authors.[11] A history suggestive of peptic ulcer warrants roentgenologic study of

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the upper gastrointestinal tract prior to their use. The presence of an ulcer contraindicates ACTH or cortisone therapy. Evidence of a healed ulcer should limit their use to cases of extreme urgency. We have seen several patients whose ulcers have perforated while under treatment with ACTH or cortisone.

The following case (case 1) demonstrates the development and perforation of a peptic ulcer in a patient who had taken large quantities of cortisone, and also the difficulty that arose from inadequate cortisone medication postoperatively. The first case was obtained from the files of Arkansas Baptist Hospital; the other cases are those of the authors.

Case Report

Case 1. J. E., a 62 year old white man, was admitted to the Arkansas Baptist Hospital on March 20, 1954, with the history of generalized abdominal pain and rigidity of several hours duration. There was no knowledge of a pre-existing peptic ulcer. An erect film of the abdomen revealed free air beneath both hemidiaphragms.

He had been taking large doses of cortisone almost daily for two years to obtain relief from *arthritis*. He was regulating his own dosage without medical supervision.

Laparotomy, at 2:00 p.m. the day of admission, showed a perforated anterior duodenal ulcer which was closed without difficulty.

He was given 25 mg. of cortisone intramuscularly two hours before operation. His postoperative course was characterized by hypotension, tachycardia, and other clinical signs of shock. Four hours after operation 25 mg. of cortisone was administered intramuscularly, with some improvement for eight hours. He again lapsed into profound shock and died 14 hours after operation. Stimulants, but no blood or additional cortisone, were administered.

At autopsy a perforated duodenal ulcer was found which was well closed. There was a marked atrophy of both adrenal cortices.

This patient's death was due, first, to the prolonged unsupervised administration of cortisone resulting in a peptic ulcer which perforated, and second, as we will point out next, to inadequate cortisone medication postoperatively.

ADRENAL CORTICAL FAILURE

Atrophy of the adrenal cortex after administration of cortisone has been observed repeatedly in both animals and man.[3, 7, 10, 14] After cortisone therapy, ACTH elicits a poor adrenal response. When cortisone therapy is withdrawn suddenly, or, postoperatively, when an increased demand is placed upon the adrenal cortex in patients who have been on cortisone, clinical evidences of adrenal insufficiency, such as weakness, hypotension and collapse are observed. A rise in eosinophil count, and lowered 17-ketosteroid levels in the urine are indicative of low adrenal cortical activity. Death from adrenal insufficiency may occur. When emergency surgery is imperative, the Thorn test[13] may be completed in four hours and will give a fairly accurate evaluation of the adrenal cortical reserve. A fall of 50 per cent or more in the circulating eosinophils four hours after ad-

ministering 25 mg. of ACTH constitutes a normal response. It should be remembered that the circulating eosinophils also will fall in response to stress or other pharmacologic agents. In the final evaluation of adrenal cortical function, changes in the excretion of urinary steroids also should be measured. Adrenal cortical atrophy has been reported as long as 12 months after cessation of cortisone therapy.

Patients who have been on cortisone therapy and are found to have a diminished adrenal cortical reserve are maintained best on intravenous cortisone in the immediate postoperative period. One hundred mg. of cortisone in 500 cc. of physiologic sodium chloride solution, administered over an eight hour period, produces a maximal metabolic effect. The dose is reduced as the patient's recovery advances.

Case Report

Case 2. J. G., a 50 year old white man, was admitted to Arkansas Baptist Hospital on March 10, 1952. A routine chest roentgenogram had shown a probable carcinoma of the posterior portion of the upper lobe of the right lung. Bronchoscopic examination failed to visualize the lesion. The patient had no pulmonary symptoms.

At thoracotomy the following day, the mass was found to be malignant and a right pneumonectomy was done. The operation was uneventful; however, upon arrival in his room, the patient's blood pressure was found to be 60/0. A liter of blood was administered rapidly, but failed to improve his condition. A continuous intravenous drip of nor-adrenalin returned the blood pressure to normal levels, but the blood pressure would drop to shock levels as soon as the nor-adrenalin was slowed or discontinued. Nor-adrenalin was given as a continuous intravenous drip for 24 hours, at which time the patient's wife volunteered the information that their family physician had been treating the patient for arthritis for several months. Hasty consultation revealed that the patient had been taking cortisone *off and on* for about six months.

He was started immediately on intravenous cortisone, 100 mg. every 8 hours for 24 hours; then 75 mg. every 8 hours for the next 24 hours; 50 mg. every 8 hours for the third day; then ACTH, 20 mg. every 6 hours for 1 week. The blood pressure stabilized immediately and remained at a satisfactory level.

ULCERATIVE COLITIS

ACTH and cortisone are of value in the treatment of patients with ulcerative colitis.[1, 4, 5, 6] In the early stages of the disease, in patients with severe symptoms, often there is an impressive increase in the sense of well-being, appetite and weight, a disappearance of fever, diminution of abdominal pain and diarrhea. Hormone therapy has reduced the mortality rate of ileostomy during an acute exacerbation of the disease. Pain, fever, tenderness and toxicity, and the physical signs of inflammation, are ameliorated by ACTH and cortisone. A mild state of euphoria is achieved. All of this may serve to mask an acute surgical condition in the abdomen.

Case Report

Case 3. Mrs. M. A. H., a 60 year old white woman, was admitted to St. Vincent's Hospital on Nov. 2, 1953. She had had a diagnosis of chronic ulcerative colitis established for many years. This had been made by roentgenogram which showed typical findings from the midtransverse colon to the anus, and by sigmoidoscopy which showed the typical hemorrhagic ulceration. She was admitted to the hospital because of a recent acute flare-up of her disease, with multiple bloody stools, anorexia and weight loss. The diagnosis was again confirmed by roentgenogram and sigmoidoscopy.

On November 2, her medical attendant began giving her ACTH gel, 60 units intramuscularly alternated with 40 units intramuscularly daily. On the morning of November 11 her intestine apparently perforated. There were no physical findings evident, and no elevation of the pulse rate or temperature. The perforation was not detected until evening rounds when the gaseous distention of the abdomen brought attention to her condition. A flat roentgenogram of the abdomen showed a large quantity of free gas. At operation she had multiple pin point perforations of the sigmoid bowel, with a generalized contamination of the abdominal cavity. The large intestine was resected from transverse colon to low sigmoid. The proximal and distal loops were brought out as colostomies. Her previous medication was continued, with the usual postoperative care, and she had an uneventful convalescence.

Pathologic examination of the excised specimen showed severe ulcerative colitis with hemorrhage. Resection of the distal portion of colon was repeatedly recommended. The patient received no further cortisone and refused any further surgery for 10 months. The colon resection was completed in September 1954, at which time postoperative hypotension was treated with cortisone.

OVERWHELMING INFECTION

ACTH and cortisone suppress the basic processes of inflammation by decreasing vascular permeability, exudation and migration of inflammatory cells.[1, 13] Thus, the classic signs of acute inflammation are reduced and the spread of microorganisms facilitated. Reparative processes are inhibited as mentioned above. The systemic manifestations, such as fever and toxemia, are decreased. All of these events produce an illusionary picture of improvement. Infection thus may be disseminated without signs of septicemia. The exacerbation of tuberculosis by cortisone is a classic example.

Case Report

Case 4. Miss E. A., a 53 year old white, single, school teacher, was seen at St. Vincent's Hospital. She gave a history of having had a reaction to penicillin, for which she received cortisone and ACTH. After about three days of this treatment she developed a large number of huge abscesses above the labia and groin and many scattered throughout the body. The one in the right groin, for instance,

was about 3 by 2 inches in diameter. Physical examination and laboratory data were within normal limits. The steroid therapy was stopped; abscesses were treated with incision and drainage, and saline soaks. They cleared promptly and have remained so until the present time.

VENOUS THROMBOSIS

Data concerning the action of ACTH and cortisone on the blood clotting mechanism are somewhat contradictory.[8, 9, 12] The concensus of opinion seems to be that these drugs may be beneficial in decreasing the clotting time, but, if given over a longer period of time, they will be associated with a higher incidence of venous thrombosis than in untreated cases. The following case seems to fall in the latter group.

Case Report

Case 5. D. D. B., a 64 year old white man, was admitted to the hospital on Oct. 5, 1953, for transfusions prior to splenectomy for a persistent pancytopenia. He had a normal cardiovascular system. The spleen was not enlarged. There was a depression of all cellular elements of the peripheral blood. The prothrombin time was 90 per cent. Capillary fragility, bleeding, clotting and clot retraction times were normal. Blood chemistries were normal.

On October 10, he was placed on oral cortisone, 25 mg. every six hours, to control severe transfusion reactions. This was continued until splenectomy was done October 22.

Postoperatively he received cortisone, 25 mg. every six hours for three days. The cortisone then was reduced 5 mg. at three day intervals until November 3, when it was discontinued.

The morning of November 4, he complained of anorexia and nausea, but was up and around the ward. The following morning he went into profound circulatory collapse. One hundred mg. of cortisone were given intravenously every eight hours. Transfusions and supportive treatment produced no improvement in the patient's condition. He complained of abdominal pain and coldness and pain in both feet. No femoral pulsations were palpable. It was thought that he probably had a mesenteric thrombosis and a saddle embolus of the aorta. He died at 5:30 p.m. on November 4, 1953. Autopsy confirmed the presence of the saddle embolus and a large thrombosis of the superior mesenteric vein.

DELAYED WOUND HEALING

ACTH and cortisone retard wound healing by an inhibitory effect on proliferation of fibroblasts and vascularization. Inflammatory elements are suppressed and slight interference with epithelization had been observed. However, serious interference with wound healing rarely occurs unless the dosage is high and prolonged (greater than 150 mg. daily).[1, 2, 13] To date we have encountered no patient who has demonstrated conclusively that cortisone has caused a delay in wound healing.

SUMMARY

The authors have observed surgical complications resulting from ACTH and cortisone medication in the following categories:

(1) Perforation of peptic ulcers aggravated or caused by cortisone;
(2) Acute adrenal cortical failure postoperatively;
(3) Perforation of a large bowel in ulcerative colitis;
(4) Overwhelming infection; and
(5) Increased incidence of venous thrombosis.

A short discussion and representative case reports are included under each heading. No instance of delayed wound healing has been observed.

REFERENCES

1. Cole, W. H., Grove, W. J., and Montgomery, M. M.: Use of ACTH and cortisone in surgery, Ann. Surg. *137:* 718 (May) 1953.
2. Cortisone and wound healing, (annotations) Lancet *1:* 15, 11 (April) p. 733, 1953.
3. Fraser, C. G., Preuss, F. S., and Bigford, W. D.: Adrenal atrophy and irreversible shock associated with cortisone therapy, J.A.M.A. *149:* 1542 (Aug. 23) 1952.
4. Kirsner, J. B., and Palmer, W. L.: Effect of corticotropin (ACTH) in chronic ulcerative colitis; observations in 40 patients, J.A.M.A. *147:* 541 (Oct. 6) 1951.
5. Kirsner, J. B., Palmer, W. L., and Klotz, A. P.: ACTH in severe chronic regional enteritis: observations in 4 patients, Gastroenterology *20:* 229 (Feb.) 1952.
6. Lahey, F. H.: Indications for surgical intervention in ulcerative colitis, Ann. Surg. *133:* 726 (May) 1951.
7. Lewis, L., Robinson, R. F., Yee, J., Hacker, L. A., and Eisen, G.: Fatal adrenal cortical insufficiency precipatated by surgery during prolonged continuous cortisone treatment, Ann. Int. Med. *39:* 116 (July) 1953.
8. Margulis, R. R.: Blood coagulation as affected by ACTH and surgical stress, S. Forum (1950) p. 507, 1951.
9. Nelson, K. O.: Influence of ACTH on venous thrombosis, S. Forum (1951) p. 316, 1952.
10. Salassa, R. M., Bennett, W. A., Keating, F. R., Jr., and Sprague, R. G.: Postoperative adrenal cortical insufficiency, J.A.M.A. *152:* 1509 (Aug. 15) 1953.
11. Smyth. G. A.: Activation of peptic ulcer during pituitary adrenocorticotropic hormone therapy; report of 3 cases, J.A.M.A. *145:* 474 (Feb. 17) 1951.
12. Stefanini, M. S., and others: Corticotropin (ACTH) and cortisone in idiopathic thrombocytopenic purpura, J.A.M.A. *149:* 647 (June 14) 1952.
13. Thorn, G. W., and others: Pharmacologic aspects of adrenocortical steroids and ACTH in man, New England J. Med. *248:* 232, 284, 323, 369, 414 (Feb. 5) 1953.
14. Ward, M. O., Stefan, S. F., and Weinbaum, J. G.: Human adrenal cortex after administration of ACTH and cortisone, Arch. Int. Med. *88:* 28 (July) 1951.

ARTERIAL HETEROGRAFTS

OBSERVATIONS ON ANIMAL EXPERIMENTS AND A REPORT OF ONE HUMAN CASE

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The clinical usefulness of preserved arterial homografts has been shown by numerous reports. The scope of surgery thereby has been enlarged so that congenital vascular defects, traumatic vessel injuries, and degenerative and occlusive disease now can be corrected by grafts. Restoration of vessel continuity incident to a deliberate radical cancer operation, including excision of a segment of a major artery, now can be done. The fate of experimental homografts following operation is fairly well documented for a five year period.[11] Further observation will determine whether histologic changes will occur or the grafts remain stable.

The procurement and preservation of arteries for clinical use becomes an important practical consideration. Frozen arterial grafts have been used successfully clinically by us for over four years. The freeze-dried method of preservation and sterilization with ethylene oxide will provide an easier and more economical method both for preservation and storage.[10] The donor source of banked preserved material usually is from individuals who died from accident. The donor should be free from malignancy, infectious diseases and be under 40 years of age. Since such rigid criteria are adhered to, the supply of arterial homografts has never satisfied the demand.

The obvious use of animal sources for grafting material has been explored. Hopfner[9] was unsuccessful in transplanting fresh arterial heterografts. Carrel[1, 2] and Guthrie[5, 6] however, were successful with their heterografting.

Gross and associates[4] reported experimental results of hog, human and baboon heterografts transplanted to the dog's abdominal aorta. These grafts were stored at temperatures of from 1 to 4 C. in a solution containing 10 per cent dog serum in a balanced salt solution. Eight animal arterial heterografts were done. One preserved human artery remained patent but became greatly dilated after two months. Two of the 4 hog heterografts were patent and in excellent condition at one and four and one-half months. The 2 remaining hog grafts showed thrombosis. The 3 baboon transplants showed intimal sclerosis, thrombosis and disintegration at four months.

Creech and co-workers[3] transplanted 56 heterografts to the carotid arteries, and thoracic and abdominal aortae of dogs. Forty-four heterografts were fixed in a 10 per cent formalin solution and 12 heterografts were preserved by freeze-drying. All of the heterografts were from hogs except 1 sheep and 3 calf arteries.

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All of the 13 transplants to the common carotid and 3 to the abdominal aorta were occluded. Nine of 18 patent aortic grafts were dilated. Gross and microscopic studies of the patent grafts showed degenerative fragmentation of the media, loss of elastic tissue, hyaline and calcium deposits and adventitial thickening.

Hufnagel and associates[10] grafted the thoracic or abdominal aorta of 23 dogs with preserved calf, pig, human, and lamb arteries. These donor heterografts were sterilized with ethylene oxide and preserved by freeze-drying. Ten calf heterografts were grafted into surgical defects of the aorta, and were observed from 8 to 402 days. All of these grafts remained patent except 1 which became partially occluded. Four pig grafts placed in the thoracic aorta remained patent from 10 to 145 days. One human graft to the abdominal aorta was patent at 153 days. Six lamb heterografts showed variable end results, and only 2 were patent. The remainder showed complications of thrombosis and hemorrhage.

Our experiments were designed to augment the observations on arterial heterografts and study species heretofore unreported.

EXPERIMENTAL METHOD

In 27 dogs surgically created defects were made in the thoracic and abdominal aortas and the brachiocephalic arteries. Restoration of the vessel defect was made with fresh or preserved heterografts. Donor arteries were obtained from human, pig, monkey, goat, and cat. Surgical excised defects of the aorta measured from 2 to 5 cm. in length. Those in the brachiocephalic were from 2 to 2.5 cm. Circumferential suture was done with a continuous no. 00000 black silk arterial suture.

Transplantation of thoracic aorta heterografts was done through a sixth rib resection thoracotomy. General body hypothermia from 28° to 30° was used to prevent distal hind limb paralysis or cardiac failure incident to acute aortic occlusion.[7] Eleven dogs survived cross-clamping, excision of the descending thoracic aorta and transplantation of the surgical aortic defect without any complications following hypothermia.

Grafting of the abdominal aorta was done through a midline incision. Surgically created defects were made between the renal arteries and the iliac bifurcation. All 11 dogs in this group survived the procedure. The brachiocephalic artery was approached through a thoracotomy incision and a surgical defect was replaced by a heterograft in 5 dogs.

The site of transplantation was determined primarily by the luminal diameter of the heterografts, every effort being made to match the diameter of the donor artery with that of the recipient. Each grafted animal received 300,000 units of penicillin daily for one week.

Frozen heterografts were preserved by immersion in a mixture of ethyl ether and solid carbon dioxide. They then were stored in an insulated dry ice chest from 7 to 180 days.

Fresh heterografts were transplanted within one hour of procurement. All irradiated grafts received an arbitrary single dose of 300r. The roentgen rays were generated by a current of 15 milliamperes having a 200 kilovolt peak. They

were filtered through 0.25 mm. of copper and 1.0 mm. of aluminum (HUL 1.2 mm. cu.). The target distance was 50 cm. Irradiation was used in an attempt to attentuate the antigenicity of the heterograft and not for sterilization. Four heterografts were transplanted within an hour after irradiation. Ten irradiated grafts were preserved by freezing from 7 to 180 days and then transplanted. Follow-up study consisted of angiography and sacrifice of the animals and histologic sectioning of the heterografts.

EXPERIMENTAL RESULTS

All of the monkey heterografts transplanted to the thoracic or abdominal aorta failed because of thrombosis (see table I). The added factor of irradiation to these heterografts did not lessen the incidence of thrombosis. These results closely parallel the observations of Gross in transplanting baboon arteries. The observation period was short, since the animals died or were killed from 28 to 80 days after operation.

Only 2 of the human heterografts remained patent. Of the remainder, 5 thrombosed and 1 ruptured at the suture line with hemorrhage (see table II). It seems remarkable that dog no. 141 observed for 48 months had a patent graft. This heterograft showed marked dilatation. The elastic fibers were fragmented and absent in many areas. The media had become very thin and replaced by scar tissue. Rupture may have ensued if the dog had been observed longer. Dog. no. 96, observed 51 months, showed an extensive adventitial fibroblastic proliferation. The media was compressed and showed hyalinization with few elastic fibers present. No dilatation was present in this human heterograft. No calcification was noted. Carrel transplanted a refrigerated human popliteal artery to a dog's abdominal aorta. Microscopic findings more than four years later showed the graft to be composed almost entirely of connective tissue with hyaline change.

TABLE I
MONKEY HETEROGRAFTS

Recipient Dog	Method of Preservation	Length of Time Vessel Preserved	Survival Post-operatively	Length of Graft	Site of Transplant	Cause of Death	Condition of Graft
#108	Frozen	45 days	60 days	3 cm.	Thoracic aorta	Died	Thrombosed
#11	Fresh	——	45 "	2.5 cm.	"	Sacrificed	Partially Thrombosed
#134	Frozen	10 days	80 "	5 cm.	"	"	"
#67	Irradiated	——	28 "	2.5 cm.	Abdominal aorta	"	Thrombosed
#100	Irradiated & Frozen	7 days	50 "	2.5 cm.	"	"	"

TABLE II

HUMAN HETEROGRAFTS

Recipient Dog	Method of Preservation	Length of Time Vessel Preserved	Survival Post-operatively	Length of Graft	Site of Transplant	Cause of Death	Condition of Graft
#141	Irradiated & Frozen	180 days	48 mos.	5 cm.	Thoracic aorta	Sacrificed	Dilated, patent
#96	"	30 "	51 "	3 cm.	"	"	No dilation, patent
#62	Fresh	——	7 days	3 cm.	"	Hemorrhage	Dehiscence proximal suture line
#64	"	——	120 "	2 cm.	Abdominal aorta	Sacrificed	Completely thrombosed
#123	Irradiated & Frozen	7 days	100 "	2.5 cm.	"	"	Thrombosed
#140	Irradiated	Transplanted same day	80 "	2.5 cm.	"	"	"
#98	Frozen	30 days	32 "	2.5 cm.	"	Undetermined	"
#124	Fresh	——	120 "	3.5 cm.	Left Brachio-cephalic a.	Sacrificed	"

TABLE III

CAT HETEROGRAFTS

Recipient Dog	Method of Preservation	Length of Time Vessels Preserved	Survival Post-operatively	Length of Graft	Site of Graft	Cause of Death	Condition of Graft
#69	Fresh	——	120 days	2.5 cm.	Left brachio-cephalic a.	Sacrificed	Patent - 90 da. Thrombosed - 120 days
#28	Irradiated & Frozen	7 days	90 "	2 cm.	"	"	Patent - 60 da. Thrombosed - 90 days
#43	Frozen	10 "	45 "	2.5 cm.	Abdominal aorta	"	Thrombosed
GOAT HETEROGRAFTS							
#33	Fresh	——	21 days	2 cm.	Left brachio-cephalic a.	Sacrificed	Thrombosed
#59	Fresh & Irradiated	——	60 "	2 cm.	"	"	"
#65	Frozen	20 days	60 "	2 cm.	Abdominal aorta	"	"
#66	Irradiated & Frozen	7 "	58 "	2.5 cm.	"	"	"

TABLE IV

PIG HETEROGRAFTS

Recipient Dog	Method of Preservation	Length of Time Vessel Preserved	Survival Post-operatively	Length of Graft	Site of Transplant	Cause of Death	Condition of Graft
#123	Fresh	——	20 days	3 cm.	Thoracic aorta	Hemorrhage	Necrosis generalized
#61	Frozen	7 days	29 "	4 cm.	"	"	Necrosis
#60	Irradiated & Frozen	14 "	48 mos.	3.5 cm.	"	Sacrificed	Patent, Good
#121	"	7 "	30 "	3 cm.	"	"	"
#97	Irradiated	Transplanted same day	20 " still alive	4 cm.	"	——	Patent, good on angiography
#82	Irradiated & Frozen	5 days	18 days	2.5 cm.	Abdominal aorta	Sacrificed	Thrombosed
#51	"	4 "	100 "	2.5 cm.	"	"	Patent, Good

Goat and cat heterografts all became thrombosed (see table III). Since the observation periods here also were short, no unusual changes were seen other than thrombosis.

Irradiated pig heterografts showed the best results (see table IV). One fresh graft and 1 frozen seven day old graft showed an extensive necrosis resulting in hemorrhagic death of these animals. Only 1 of 5 irradiated grafts thrombosed. The histologic changes observed in the successful irradiated pig heterografts included a degeneration of the media with hyalinization of the media. Dog no. 60, killed at 48 months, showed areas of calcification. The media became thinned and more dense with fragmentation and disappearance of the elastic fibers.

CASE REPORT

A 64 year old white man was admitted to the University of Kansas Medical Center in December 1953. He noticed a pulsating mass developing in his right groin. The onset was gradual but progressive over a three year period. In February 1953 he developed sudden pain and numbness in the sole and toes of his right foot. This episode was attributed to an arterial embolus probably arising from the femoral aneurysm. These symptoms abated after several months of bed rest. In May 1953 again he developed pain in the right foot, swelling of the right leg and hemoptysis. He was hospitalized for two months and the right leg was treated with paravertebral blocks. Since this last episode he had increasing ambulatory activity but had exercise pain in the lower calf. His activity thereby was limited to walking a block or so with periods of rest. He also had an attack of coronary occlusion in 1941 and in 1952. On physical examination he showed

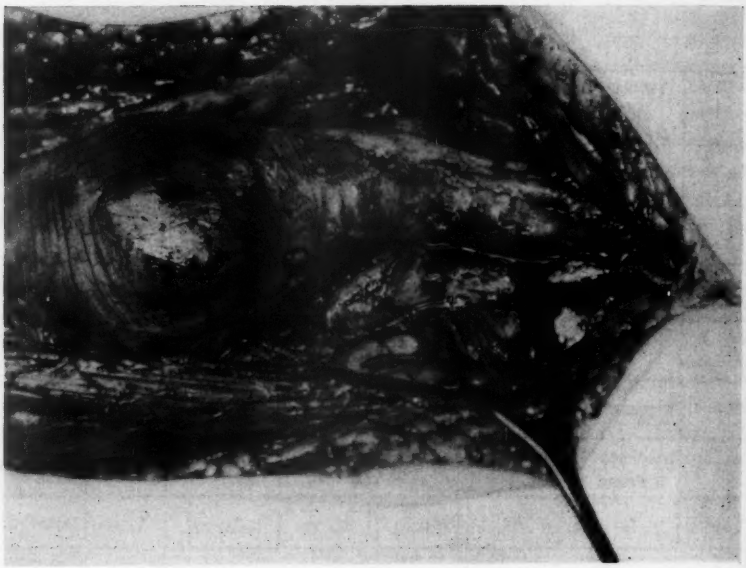

FIG. 1. Arteriosclerotic aneurysm of right iliac and femoral artery in a patient 64 years of age.

evidence of generalized arteriosclerosis. A pulsating aneurysm measuring 6 by 4 cm. in diameter was present in the right groin. It extended proximally beneath the inguinal ligament. Bilateral pulses could be felt in the popliteal and dorsalis pedis arteries.

On Jan. 1, 1954 the right femoral aneurysm was operated upon. A retroperitoneal approach was made proximally, dividing the inguinal ligament and exposing the iliac artery retroperitoneally (fig. 1). The artery was occluded proximal and distal to the aneurysm with Blalock clamps. Heparin solution was injected into the artery above and below the clamps. A segment of the iliac and distal femoral artery 22 cm. long, was resected. The profunda femoris artery was occluded and no perforating bleeding vessels were seen in the bed of the aneurysm after excision. Because of the length of the surgical defect preserved pig arteries were sutured together in order to reconstruct the iliac and femoral arteries (fig. 2). These pig heterografts were irradiated and then frozen for 24 days in a manner as previously described. Arterial anastomoses were done with interrupted and continuous no. 00000 black arterial silk suture. The inguinal ligament was resutured and a layered closure of the wound was made. Pathologic examination of the resected specimen showed an arteriosclerotic aneurysm with an organizing thrombus.

The patient was ambulatory on the fourth postoperative day. This patient is now active and ambulant nine months following heterografting of the iliac and femoral arteries. Exercise pain is gone and he no longer needs to rest while walk-

FIG. 2. Repair of iliac and femoral arteries following excision of aneurysm shown in figure 1. Irradiated pig heterograft 22 cm. in length transplanted to surgical defect. This heterograft was utilized because no homografts were available at this time.

ing. He states he can walk as far as he wishes without symptoms in a normal days activities. Angiography of the grafted segment shows no dilatation.

DISCUSSION

Clinical heterografting has been reported by Leger and Oudot.[13] A 2 cm. segment of femoral artery was replaced with a dog's artery preserved for four days. Angiography one month later showed good patency of the graft. Length of further follow-up was not stated.

Hufnagel and associates,[10] encouraged by experimental results with freeze-dried calf and pig heterografts, reported their use in 4 human beings. The common iliac was replaced with segments of calf and pig heterografts. Patency was demonstrated by angiography six months following grafting. Three further cases are cited in which the segments of the radial, popliteal and iliac arteries were grafted with calf heterografts.

Creech and associates[3] report transplantation of a freeze-dried pig graft to the left iliac artery of a patient. Dilatation of the heterograft was seen on angiography at five months.

Hui and co-workers[12] used high intensity electrons to effect sterilization of aortic homografts. Irradiated grafts were treated by means of a 3 million volt Capacitron. After irradiation the grafts were frozen quickly in liquid nitrogen and then stored at 70 C. until used. Twenty-two dogs were grafted with irradiated aortic segments and satisfactory functional results were noted in 16 animals.

Observations were from one day to six months. Microscopic findings were like those observed in sterile nonirradiated grafts. However, more striking degenerative changes were seen in the media of irradiated grafts implanted after two months. At four months unusually heavy calcium deposits were present, becoming more pronounced at six months.

Irradiation was selected as an experimental method of attenuating any antigenic dissimilarity of the heterografts. Successful homografting by the use of irradiated donor skin to normal CFW strains of mice would seem to substantiate the above theory.[8]

Hufnagel's success in transplanting freeze-dried pig and calf heterografts may be attributed to some change in the immune response of the donor graft to the recipient. Ethylene oxide is a fat solvent also and removal of fatty compounds might play an important role in the behavior and fat of heterografts so treated.[11] However, Hufnagel and associates point out that if the drying process is not carried out properly that undesirable histologic changes are present in such an implanted heterograft.

Creech and co-workers report divergent results using freeze-dried heterografts.

Further experiments now are in progress in which the irradiating dosage is being varied. Irradiated freeze-dried grafts also are being studied for a comparison. Electrophoretic studies on the serum of the heterografted dogs also are being done. Efforts also are being made to determine the presence of any circulatory antibodies.

SUMMARY

A successful transplant of an irradiated pig heterograft to the iliac and femoral artery of a human being is reported. This graft is functioning well nine months after implant. All of the monkey, goat, and cat heterografts thrombosed regardless of the manner of preservation. Human heterografts generally failed because of thrombosis and necrosis. The patent human heterografts showed dilatation and undesirable histologic change. Fresh heterografts in all the species studied failed because of necrosis or thrombosis.

Irradiated pig heterografts yielded the best results. Although these grafts remained patent up to 48 months, the histologic degeneration seemed further advanced as compared to a homograft observed for a similar length of time.

Longer periods of observation and study are necessary to predict the ultimate fate not only of heterografts but also of homografts.

REFERENCES

1. Carrel, A.: Heterotransplantation of blood vessels preserved in cold storage, J. Exper. Med. *9:* 226, 1907.
2. Carrel, A.: Ultimate results of aortic transplantation, J. Exper. Med. *15:* 389, 1912.
3. Creech, O., DeBakey, M. E., Self, M., and Halpert, B.: Fate of heterologous arterial grafts; experimental study, Surgery *36:* 431 (Sept.) 1954.
4. Gross, R. E., Bill, A. H., and Peirce, E. C.: Methods for preservation and transplantation of arterial grafts, Surg., Gynec. & Obst. *88:* 689 (June) 1949.
5. Guthrie, C. C.: Heterotransplantation of blood vessels, Am. J. Physiol. *19:* 428, 1907.
6. Guthrie, C. C.: Survival of engrafted tissues III Blood vessels, Heart *2:* 155, 1910.
7. Hardin, C. A., Reissmann, K., and Diamond, E. G.: Use of hypothermia in resection and homologous graft replacement of thoracic aorta, Ann. Surg. (*140:* 720, 1954).

8. HARDIN, C. A., and WERDER, A. A.: Effect of total body irradiation on survival of homologous skin grafts on CFW mice, Plastic & Reconst. Surg. *13:* 40 (Jan.) 1954.
9. HOPFNER, E.: Ueber Gefässtransplantation used replantation von amputierten extremitoten, Arch. klin, Chir. *70:* 417, 1903.
10. HUFNAGEL, C. A., RABIL, P. J., AND REED, L.: Method for preservation of arterial homo- and heterografts, S. Forum (1952) p. 162, 1953.
11. HUFNAGEL, C. A.: Transplantation of blood vessels, preservation and transplantation of normal tissues, Ciba Foundation Symposium, Boston, Little Brown and Company, p. 169, 1954.
12. HUI, K. K. L., AND OTHERS: Early results of experimental studies of action of high intensity electrons on aortic homografts, S. Forum (1951) p. 255, 1952.
13. LEGER, L., AND OUDOT, J.: Greffe arterielle hétérogéne, Mem. Acad. chir. *76:* 728 (June 28 to July 5) 1950.

AN IMPROVED METHOD FOR ARCH-LIKE GASTRIC RESECTION

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In an earlier report, the author[6] described the development of a new curved sewing clamp and its preliminary use in an improved method for arch-like gastric resection.[1, 3, 4] With this instrument, the lesser curvature of the stomach can be resected and sutured in an arch-like manner almost parallel to the greater curvature so that a tube-like structure is formed and the remaining portions of the stomach elongated, thus permitting gastroduodenal anastomosis without excess tension at the suture line. It should be emphasized that this method enables the operator to do an accurate arch-like type of radical resection and provides a means for rapid, aseptic, complete suturing of the gastric wall (figs. 1, 2).

Two sewing clamps are used for this type of reconstruction. One is a short, straight sewing clamp for the partial bisection of the stomach body from the greater curvature side, and the other is a curved one for the arch-like resection of the lesser curvature. These clamps are identical except for the curve of the latter. The anterior jaw of the clamp is equipped with two rows of regular spaces which contain clips. After insertion of the metal clips, the clamp is applied to the prearranged portion of the stomach and then is tightly closed by firmly grasping the handle. The interpositor is removed and the pincer forceps then are applied to the indentations. This maneuver crushes the stomach and pushes down the clips which suture the stomach wall. By this single procedure the stomach wall is closed with clips and can be bisected without hemorrhage.

OPERATIVE TECHNIC

The patient is prepared in the usual manner for major gastric surgery. Prior to operation, a narrow double lumen rubber tube is inserted into the stomach through the nose. The upper abdomen is opened by a median incision and the stomach is mobilized in the usual manner. The gastrocolic omentum is dissected towards the pylorus and the fundus to the point where resection is anticipated. The gastroepiploic artery is ligated and the duodenum is mobilized. The same procedures are completed with the lesser omentum and the right gastric artery is ligated. Following adequate mobilization of the duodenum, two Kocher forceps are applied to the duodenum where transsection is anticipated, and the duodenum then is divided between the forceps. The stomach is pulled forward and the left gastric artery is ligated high and divided. Thus, complete dissection of the lesser omentum results in free mobilization of the lesser curvature.

A short, straight sewing clamp then is applied transversely to the half of the stomach body from the greater curvature side, a little distal to the anticipated resection line (fig. 3). Following transverse partial bisection of the greater curvature side of the stomach, a curved sewing clamp is applied and a portion of

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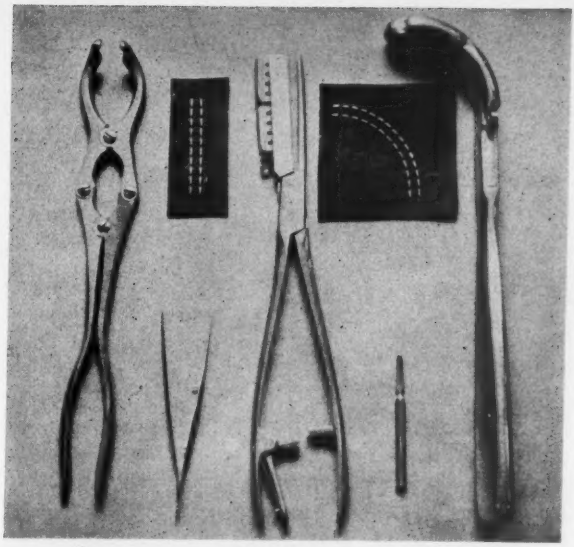

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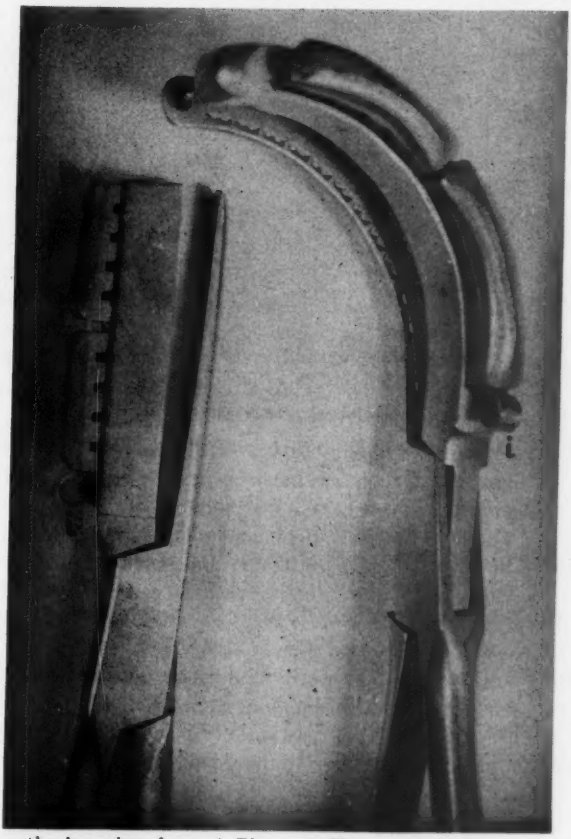

FIG. 1. The author's sewing clamp. 1, Pincer; 2, Short straight clamp; 3, Curved clamp; i, Interpositor.

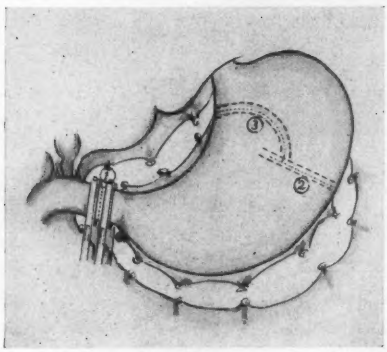

FIG. 2

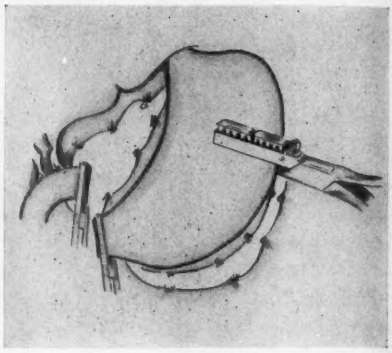

FIG. 3

FIG. 2. The broken line and the numbers show the outline of the area of resection.
FIG. 3. The half of the stomach body from the greater curvature side is transversely bisected with the use of the author's short straight clamp.

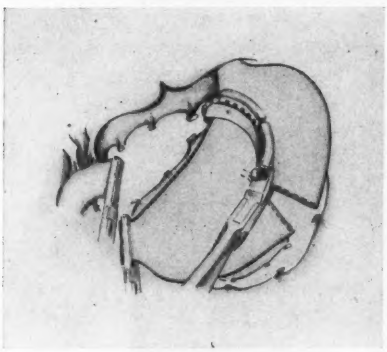

FIG. 4

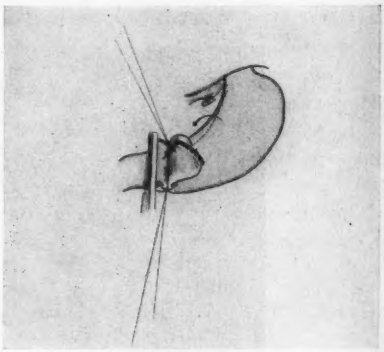

FIG. 5

FIG. 4. The lesser curvature side is resected in an arch-like manner nearly parallel to the greater curvature with the use of the curved clamp.
FIG. 5. The sutured line of the lesser curvature side of the new stoma is rotated 45 to 90° posteriorly and the gastroduodenal anastomosis is completed.

the lesser curvature side, extending to the cardia, is resected in an arch-like manner (fig. 4). This line of resection must be parallel to the greater curvature and the lumen thus formed must have a diameter a little larger than that of the duodenal stump. This is accomplished by controlling the position of the sewing clamp and exerting traction on the stomach anteriorly and inferiorly. By the use of these two clamps, the procedure of resection can be done easily and aseptically, and the new pouch thus formed is quite mobile and elongated. The line of resection on the lesser curvature then is closed with interrupted silk sutures and the new pouch is rotated posteriorly 45 degrees to 90 degrees (after Kirschner), and in this position a stomach clamp is applied to the new pouch to prevent hemorrhage when completing the anastomosis. It is advisable to increase the degree of posterior rotation according to the size of the resected area—the larger

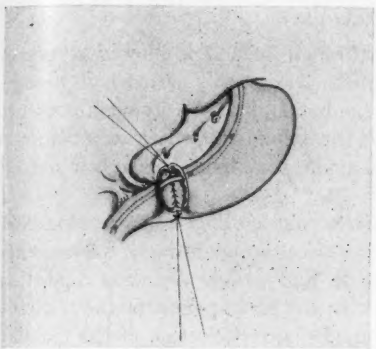

FIG. 6

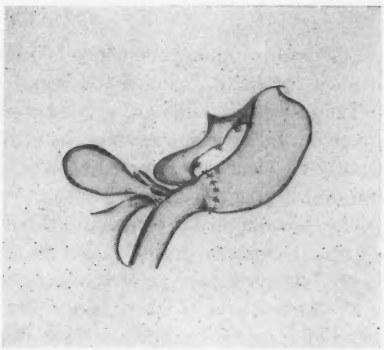

FIG. 7

FIG. 6. When the posterior interrupted sutures have been completed, the double lumen tube is passed into the duodenum.

FIG. 7. The anastomosis is completed. Note the dissection of adhesions at the convexity of the duodenum.

the size, the greater the degree of rotation. The gastric and duodenal stumps then are brought into position for anastomosis, and an end to end anastomosis is made. If it appears that the lumen of the tract is going to be narrow, it is advisable to resect the stump obliquely for this will allow a larger passageway. Two stay sutures, one at the superior and the other at the inferior aspects of the anastomosis line, are inserted deeply and both sides of the duodenal and gastric stumps are sutured tightly (figs. 5, 6). The author usually does not apply the posterior sero-muscular sutures, but if it is necessary, a few sutures may be used, and one of them can be inserted transversely at the site of the suture line on the lesser curvature. The lumens then are opened on both sides. When the posterior interrupted sutures have been placed, the double lumen tube, which is in the stomach, is passed into the duodenum (fig. 6). Following this, the anterior reconstruction of the anastomosis usually is completed with interrupted sutures (fig. 7). If there is still excess tension at the anastomosis site, it is due probably to abnormal adhesions in the duodenal wall. Dissection of adhesions at the convexity of the duodenum or adhesions remaining in the gastric bed will reduce this tension.

CASE REPORTS

This type of procedure has been used on 100 patients to date and in most of them an estimated two-thirds or subtotal gastric resection was done. Of these 100 patients, 6 were operated upon because of repeated hemorrhage; 2 for acute perforation; and 18 showed penetrating gastric ulcers. Severe obstructions were seen in 6 patients, 1 of which presented a typical hour-glass stomach. The remaining 65 patients were of the ulcer type who had not responded to intensive medical treatment and who persistently complained of pain and gastric distress over a prolonged period of time.

POSTOPERATIVE COURSE

The double lumen tube which is inserted through the nose is allowed to remain in place for 48 to 72 hours following operation. During this period high protein and high caloric fluids are given through one lumen, and the stomach contents are aspirated continuously via the other. On the second day, the gastric contents usually are clear or are mixed with bile, and at this time the tube can be removed and oral intake begun.

The postoperative course in these patients was studied roentgenologically (fig. 8). Roentgenograms taken about two weeks postoperatively infrequently showed delayed passage of the gastric contents and a slight degree of dilatation of the new stoma. Such patients were seen in the early period of the author's series of cases and may have been due to inadequate perfection of the technic. Within four weeks these symptoms had disappeared completely and the barium meal passed rapidly through the anastomosis into the intestine a few minutes after it had reached the stomach. The stomach, which is now tube-shaped, appeared to have restored tonus. Edema of the anastomosis site disappeared in all patients, and no cases of stricture were evident even in those who showed a tendency towards stasis of the gastric contents. Some patients maintained a rather rapid emptying time, but none gave evidence of the postgastrectomized syndrome.

RESULTS

There were no direct mortalities after this operation. The gastric juice of patients was examined preoperatively and postoperatively. Half of the patients

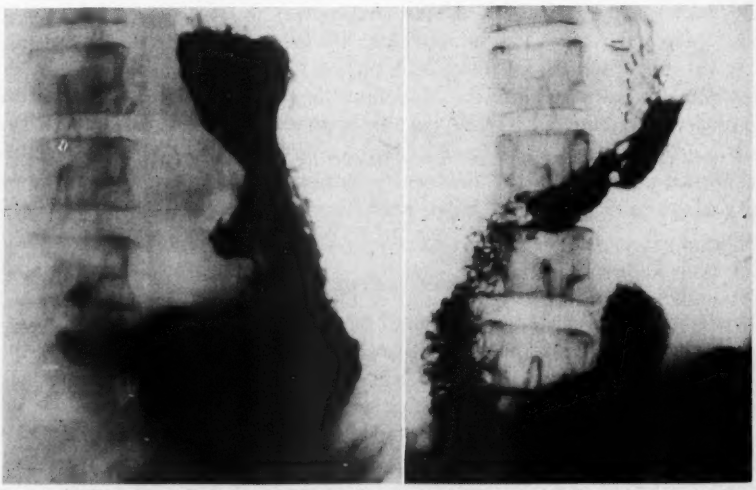

FIG. 8. A; Preoperative roentgenogram showing a large penetrating ulcer in the lesser curvature. The patient, aged 44, female. B; Roentgenogram of the same patient taken two months after operation.

TABLE I

Preoperative and Postoperative Free Hydrochloric Acid

Case	Preoperatively		Two to Three Weeks After Operation		Six Months to Two Years After Operation	
	Before Test	After Test	Before Test	After Test	Before Test	After Test
M. Y.	36	76	10	25	0	0
M. S.	8	60	0	0	—	—
K. I.	0	55	0	10	—	—
M. M.	0	28	0	0	0	0
U. T.	0	70	0	0	—	—
F. A.*	30	34	0	0	0	0
K. Z.*	5	80	—	—	0	0
M. K.	12	24	—	—	0	0
T. K.	9	42	0	0	0	0
T. S.*	0	5	0	0	0	0
S. S.	44	24	0	0	0	0
U. E.	0	5	0	0	—	—
F. S.*	0	32	0	11	0	0
S. E.	10	14	0	6	—	—
S. Y.	—	43	0	0	0	0
B. M.	6	0	0	0	0	0
K. S.	40	68	0	0	—	—
Y. M.*	34	56	0	0	—	—
K. I.	60	96	0	0	—	—
K. O.	0	0	0	0	—	—
I. Y.*	0	26	—	—	0	0
S. R.*	0	22	0	12	0	10
T. I.	22	60	0	0	—	—
K. I.	40	22	0	0	—	—
M. I.	14	42	0	0	—	—
K. A.	50	40	0	0	—	—
K. K.	25	55	—	—	0	0
T. U.*	44	62	0	0	—	—
S. N.	38	95	16	30	—	—
K. K.	6	14	—	—	0	0

NOTE: The number shows the highest free acid value during the test. The cases marked * were examined with Histamin test; the remaining cases with Katsch-Kalk test.

had a high free acidity preoperatively, and the gastric juice examined about two weeks postoperatively generally showed an absence or a low value of free acid, but there was individual variation and the findings were labile. After six months to two years, all patients examined except 1 showed an absence of free acid (table I).

DISCUSSION

In order to obtain effective results in the surgical treatment of peptic ulcer, the gastric resection must be radical, and the reconstruction must be physiologic. At the same time, the procedure must be safe and easy. By the method here described, a radical resection of the stomach can be done, and it will include

the entire lesser curvature where most ulcers occur. Not only does this method remove the tissue most predisposed to the ulcer formation, but the reconstruction is physiologic in that it follows the normal anatomic pathway of the gastric contents. Also, the absence of free hydrochloric acid, which is indispensable for the cure of peptic ulcer, is achieved in almost all patients. The tension placed at the anastomosis is lessened due to mobilization and elongation of the lesser curvature by the arch-like resection. With the use of the sewing clamps, the arch-like resection of the lesser curvature can be done with safety and ease. The weak point at the triangular portion is avoided by the posterior rotation of the suture line of the newly resected lesser curvature side, and this posterior rotation maintains good function of the new stoma because of its natural position. Stricture at the anastomosis, frequently seen as one of the defects of the Billroth I operation, is avoided by the use of interrupted sutures and other technics described above.

According to Takahashi,[5] 21 of 28 patients who had the Billroth I type of resections (Ohtsuki's Clinic, Tokyo) showed periodic emptying through the anastomosis, while 41 of 66 patients of the Billroth II showed continuous emptying. Although some patients, even of the Billroth I type, had a tendency toward rapid emptying, the so-called postgastrectomy syndrome never was seen. However, there were a few patients with the Billroth II operation who complained of the *dumping* syndrome to a slight degree. As previously mentioned, in a majority of patients treated by the author's method, the barium meal empties through the anastomosis synchronously with the intestinal peristalsis. Although a few patients maintained a rather rapid emptying time, none developed the dumping syndrome.

This method is considered to be more physiologic than the Billroth II, and it has a wider use than the ordinary Billroth I because it permits a radical resection if necessary. This method of arch-like gastric resection with the clamp also is convenient when doing the Billroth II. For this reason, it is quite natural that this method has been employed for the treatment of malignancy. At present the series of gastric carcinomas is not large enough to draw any accurate conclusions. They will be reported at a later time.

CONCLUSIONS

A modification of the Billroth I operation with an arch-like gastric resection employing new clamps, is described. Results with 100 gastroduodenal ulcer patients upon whom this type of resection and reconstruction has been done are presented with their follow-up histories.

This method is believed to be an improvement over the other technics currently employed because it is a simple, rapid and safe surgical procedure which follows physiologic principles. It has a wide application and will permit radical resection to be done quickly and easily.

REFERENCES

1. Kirschner, M.: Allgemeine u. spezielle chirurgische Operationslehre, Siebenter Band Teil I, 1951, (Springer-Verlag).

2. OHTSUKI, K.: New simplified gastro-intestinal sewing clamp with metal clips, J. Med. Instr. (Japan) *18:* ⁂1, 1, 1941.
3. RIEDER, W.: Eine neue Magenklemme zur Resektion cardianaher Ulcera, Zentralb. f. Chir. *59:* 2095 (Aug. 27) 1932.
4. SHOEMAKER, quoted by GRAHAM, R. R.: Technical surgical procedures for gastric and duodenal ulcer, Surg., Gynec. & Obst. *66:* 269 (Feb.) 1938.
5. TAKAHASHI, H.: Studies on passage of resected stomach, J. Japanese Surg. Soc. *51:* ⁂10, 608, 1951.
6. YAMAGISHI, M. An improved method for arch-like gastric resection, Yokohama Med. Bull. (Japan) *4:* ⁂ 6, 365, 1953.

PERFORATION OF DUODENAL STUMP FOUR MONTHS AFTER SUBTOTAL GASTRECTOMY

REPORT OF A CASE

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Perforation of a duodenal stump is a serious complication of the early postoperative period following gastric resection. Usually such perforation occurs within the first two weeks after the operation. Perforation occurring months after the gastric resection is unusual, only a few cases having been reported in the literature. Goodall, Hindman and Heimlich[1] reported a case occurring 5½ months after subtotal gastrectomy, and Sanchez[2] described a case occurring after a period of 10½ months. We are presenting a case report of perforation of the duodenal stump 4 months after gastrectomy.

CASE REPORT

A white man aged 29, was admitted to the Indianapolis Veterans Administration Hospital in January 1953, because of pyloric obstruction and intractable symptoms due to duodenal ulcer. There had been also several episodes of massive melena in the months prior to admission. Subtotal gastrectomy with an antecolic gastrojejunal anastomosis was done. The duodenal stump was closed with a layer of chromic catgut sutures and a layer of inverting interrupted silk sutures. It was then turned into the head of the pancreas with a third layer of interrupted silk sutures. He recovered without difficulty and was well until April 30, 1953, when he developed acute cramping epigastric pain and nausea one-half hour after eating his evening meal. On admission to the hospital soon after the onset of the pain the patient was pale and cold with a temperature of 96 F., a pulse of 66 and a blood pressure of 135/95. Mild generalized abdominal tenderness was present and a rather ill-defined mass, just to the left of the midline above the umbilicus, was noted. A white blood count was 15,000 per cu. mm. with 88 per cent neutrophils. When we first saw the patient about 12 hours after admission, generalized abdominal tenderness and rigidity were present, especially over the right side of the abdomen. He was acutely ill with increased subjective pain. Immediate abdominal exploration was done using a muscle-splitting incision in the right lower quadrant. Much yellow fluid was encountered in the peritoneal cavity and the grossly normal appendix was not removed. The incision was closed and the peritoneal cavity was re-entered through the old gastrectomy incision. The

From the Department of Surgery, Veterans Administration Hospital and The Indiana University Medical Center. Reviewed by the Dean's Committee and published with the approval of The Chief Medical Director. The statements and conclusions published are a result of the authors' study and do not necessarily reflect the opinion or policy of The Veterans Administration.

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gastroenterostomy was normal in all respects except the proximal jejunal loop which was congested and dilated. However, a severe peritonitis was present in the upper abdomen. The duodenal stump was exposed and active leaking of bile-stained fluid from a small perforation in the suture line was discovered. The perforation was closed with mattress sutures and the area was drained. The patient recovered. Several months later he was admitted again and was operated upon for small bowel obstruction due to adhesions. He has been well since the last operation.

DISCUSSION

The similarity between our case and the case reported by Goodall is rather striking. In each patient the illness began with epigastric pain shortly after eating. A mass, which later disappeared, was found in the same location above the umbilicus. In neither was the clinical picture of a perforated viscus evident on first examination, delaying early operative intervention. At operation, in each patient, the proximal loop of jejunum of the gastro-jejunostomy was found to be dilated and congested, although no evidence of obstruction was found to account for the dilatation. Goodall thought that transient obstruction of the proximal jejunal loop, possibly due to angulation at the gastrojejunostomy stoma, may have initiated the difficulty. He had histologic evidence of a foreign body reaction and a sinus tract at the site of perforation and believed that this might represent a point of least resistance following obstruction and dilatation of the jejunal loop. The sequence of events in our patient may have been the same, although we have no evidence of a sinus tract other than the fact that the perforation occurred in the suture line of the duodenal stump where a foreign body reaction most likely would be encountered. A reconstruction of the clinical course, rather similar to that presented by Goodall, is as follows: After eating, transient obstruction occurred at the gastrojejunostomy stoma with dilatation of the proximal jejunal loop. This could have accounted for the initial symptoms of cramping pain and nausea. The mass which was found on abdominal examination probably was the dilated jejunal loop, which disappeared when perforation of the duodenum at the stump suture line occurred. The clinical picture of a perforated viscus developed rapidly with leakage of bile and intestinal contents into the peritoneal cavity.

CONCLUSION

Due to the serious nature of the biliary peritonitis caused by this late complication of subtotal gastrectomy, consideration of the possibility of duodenal stump perforation is indicated in the postgastrectomy patient presenting acute abdominal symptoms, even if some months have passed since the original operation.

REFERENCES

1. Goodall, R. E., Hindman, R., and Heimlick, H.: Perforation of duodenal stump 5½ months after subtotal gastrectomy, Surgery *30:* 701 (Oct.) 1951.
2. Sanchez, Zinny J.: Ruptura del Munon duodenal. Complicacion alejada de la gastrectomia, Bol. y trab., Soc. argent. de cirujanos *5:* 1124, 1944.

NEUROFIBROMATOSIS PRODUCING PERSISTENT NECK PAIN AND PARALYSIS OF RIGHT ARM FOLLOWING PRE-ECLAMPSIA

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Multiple neurofibromatosis or von Recklinghausen's disease may present itself in many weird ways. It may cause a localized progressive paralysis or it may be associated with almost any pathologic condition. It may remain silent in its progress until another illness appears and then loudly announce its presence. The case under consideration is one in which the neurofibroma remained relatively silent until the patient became seriously ill with pre-eclampsia.

CASE REPORT

L. B. a 19 year old, unmarried, white, gravida I, para 0 female had been admitted to Florence Crittenden Home on June 21, 1953 for complications of pregnancy consisting of shooting pains in her right shoulder, arm, and posterior neck. The blood pressure on admission was 120/80; the urine was negative for albumin. The condition was diagnosed as myositis and treated with heat, tolserol, aspirin and codeine without relief. A week later, the blood pressure rose to 140/104, and albuminuria was noted. All medications were discontinued, and a pre-eclamptic regime was instituted. Her blood pressure remained elevated; a 4 plus albuminuria persisted, and a 1 plus pitting edema of the legs appeared. The blood pressure rose to 160/110 on July 1. Because of this condition and the presence of abdominal pain, the patient was transferred to the Colorado General Hospital.

On July 4, a 1250 gram baby was delivered and the symptoms of pre-eclampsia disappeared. The baby died July 5, 1953.

The patient had had mumps on March 24, 1953 with marked parotid enlargement for the duration of a week, followed in two weeks by neck pain and stiffness, general malaise, weakness in the right arm, and progressive numbness and dead feeling in the right arm and hand. These symptoms were relatively mild at the onset but had progressed on admission to the point that her right arm and hand were useless and the pain in the posterior right neck was debilitating. There were no changes in voice, ability to swallow, vision, hearing, or facial sensation.

Physical examination revealed a well developed, well nourished young girl who was lethargic but fairly cooperative and who complained of severe pain in her neck and left arm, and of inability to move her right arm. A significant finding was that of a grade II systolic murmur at the heart apex. There was marked restriction of neck movements; the patient held her head slightly turned to the right in mild flexion. Café-au-lait spots were noted over her arms and back.

Neurologic consultation on July 8, 1953 revealed astereognosis, absent posi-

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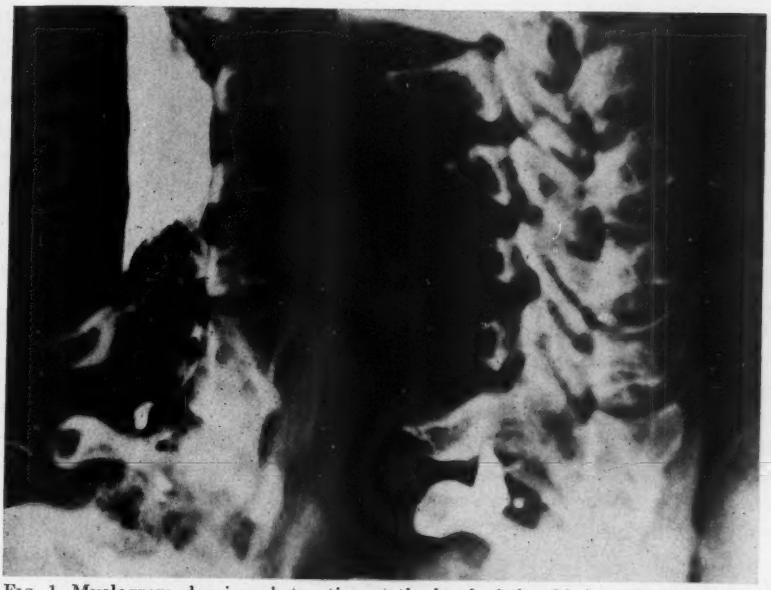

FIG. 1. Myelogram showing obstruction at the level of the third cervical vertebra.

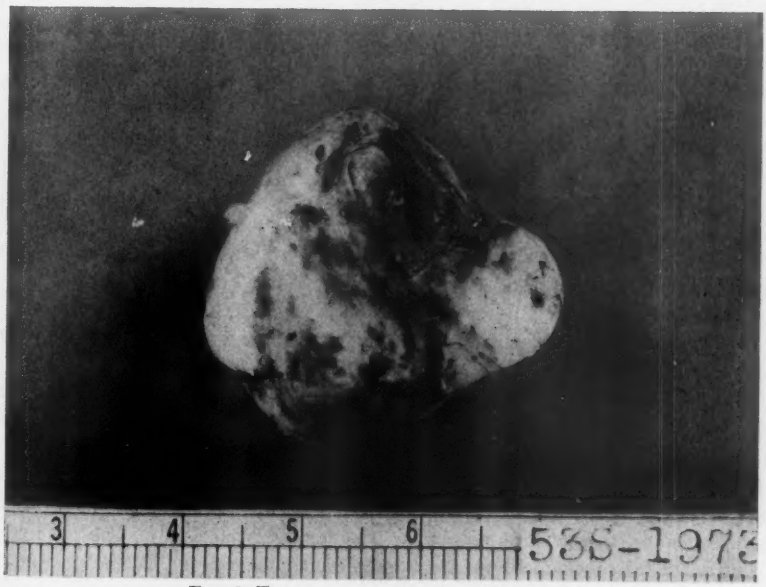

FIG. 2. Tumor removed at time of surgery.

tion-sense, increased deep tendon reflexes, and Chaddock's sign on the right; right hemiparesis which was maximal in the right arm; diminished two point discrimination and loss of vibratory sensation in both the right and left arms; decreased abdominal reflexes; hyperesthesia of the area described by the second cervical dermatome through the second thoracic dermatome on the right and hypesthesia in the area covered by the second, third, and fourth cervical dermatomes on the left. The resulting impression was a high cervical cord tumor or cervical arachnoiditis. The possibility of hysteria was suggested in view of the tearful, depressed picture and the feelings of guilt because of the death of the baby. The fact that the patient was emotionally disturbed was obvious, but this was not a complete explanation for the pain or the neurologic findings.

On July 31, 1953, a diagnosis of cervical cord tumor was made. In view of the previously negative cervical myelogram, a pneumoencephalogram was considered. This also was unsuccessful in locating the tumor, but residual pantopaque from the previous myelogram showed obstruction at the level of the third cervical vertebra (Fig. 1). On Aug. 18, 1953, cervical laminectomy (by R.M.S.) showed a large neurofibroma of the second cervical nerve root measuring 3 by 2.8 by by 2 cm. (Fig. 2). The tumor was removed and the spinal cord was restored to its normal position. Pertinent laboratory data are summarized as follows:

1. Persistent 1 to 4 plus albuminuria: Multiple spinal punctures showed elevated protein, normal manometrics, negative Kolmer, gold curve normal, and cultures negative.

2. Roentgenologic data: July 7, 1953 Cervical spine—negative; July 8, 1953 Cervical spine—negative; July 17, 1953 Cervical myelogram—negative; Aug. 6, 1953 Pneumoencephalogram—negative; residual pantopaque from myelogram—obstruction level third cervical vertebra revealed.

3. Review of roentgenograms: On Aug. 17, 1953 the right intervertebral foramina between second and third cervical vertebrae was enlarged more than two times its normal size, measuring 19 by 12 mm. Anteroposterior views made at time the myelogram was made, and lateral views made at the time the pneumoencephalogram was made, (both made with patient in the marked Trendelenburg position) showed a partial obstruction at the level of the third cervical vertebra. It was our impression that there was a neurofibroma at the level between the second and third cervical vertebrae enlarging the right intervertebral foramen.

4. Pathology report: Encapsulated benign neurolemmoma of a spinal nerve root (Fig. 3).

The postoperative course was uneventful, and the remainder of the hospital course was concerned with physical therapy and rehabilitation. Improvement of muscle function was progressive, with marked restoration of function.

On June 15, 1954, 10 months after operation, this patient was readmitted to the Colorado General Hospital with a complaint of a painful mass in the right supraclavicular region. This new mass was removed surgically. The pathology report again was benign diffuse neurolemmoma.

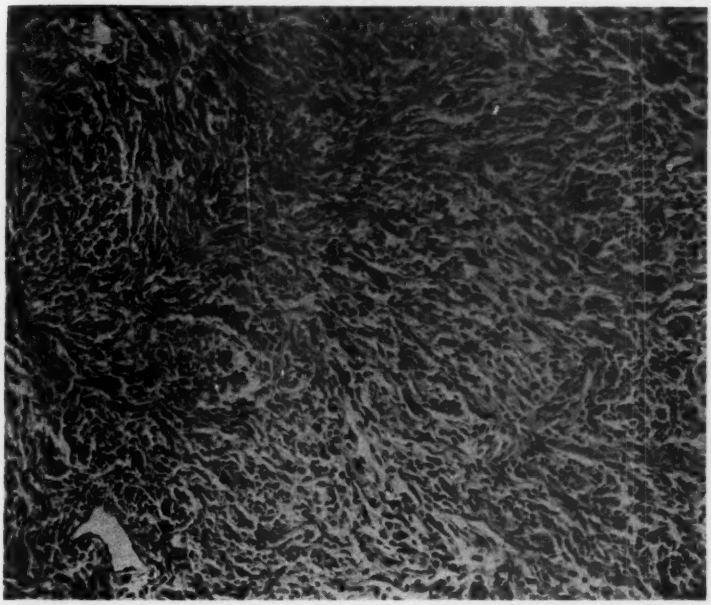

FIG. 3. Microscopic section of tumor showing palisading of nuclei which is typical of neurofibroma.

SUMMARY

This is a case report of a patient with neurofibromatosis, who developed a right-sided paralysis due to pressure of a tumor on the cervical spinal cord. This pressure-producing tumor was removed surgically with excellent restoration of muscle function. Ten months later, the patient was readmitted for excision of a painful neurolemmoma in the right supraclavicular region. The prognosis for cure of this disease is poor; the future holds the probability of multiple excisions of bothersome tumors involving any or all of the peripheral nerves.

SUCCESSFUL REMOVAL OF A NONCHROMAFFIN PARAGANGLIOMA OF A VAGUS NERVE

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Within the last 10 years great interest has been manifested in neoplasms composed of tissue which closely resembles that of the carotid body. The consensus appears to be that the cells in question belong to a system of chemoregulators that are widespread within the human organism. Neoplastic growths arising in these small ganglia are being recognized with increasing frequency. They have been described in and around the middle ear, jugular foramen, within the vagus nerve, the carotid body, about the arch of the aorta in the chest and abdomen.

The total number of cases so far reported in all locations probably does not exceed 400 which readily indicates their relative infrequency. In the vast majority of patients the tumor is benign. Often it has a propensity for invading contiguous tissues but rarely has conclusive evidence been reported that distant metastases have occurred.

Tumors of this variety arising from the intravagal ganglia are very uncommon and most of those reported have involved so many intracranial structures that surgical removal has not been possible. Should they arise in the cervical ganglion of the vagus nerve, however, excision is possible and such a case is reported.

CASE REPORT

A 64 year old woman was seen originally in the otorhinolaryngology department of this clinic with the complaint of a mass in the right side of her throat. Her difficulties had begun about six months before presenting herself here when she became aware of a *funny feeling* in the right side of her throat. Shortly after this she noticed a mass near the right tonsil. Whether it pulsated at this time is not known. Several physicians were consulted and one removed tissue for biopsy. Fortunately he secured mucous membrane only. It was assumed that the tonsillar area was infected. Antibiotics and radiation therapy were administered without results either to the size of the mass or to the patient's symptoms.

When examined, it was plain that the tonsillar area on the right was pushed medially by a firm, nontender mass beneath it. The whole area was visibly pulsating synchronously with the pulse rate. A systolic bruit could be heard over the area. Nothing with certainty could be felt externally in the neck and on clinical grounds it was assumed the patient had an aneurysm of the internal carotid artery. There were no neurologic findings at this time.

A carotid arteriogram was done and a vascular, roughly spherical tumor about the size of a small lemon was outlined, well above the carotid bifurcation, close to the base of the skull. The internal carotid artery was displaced forward (fig. 1). It now was evident what was producing the pulsating mass in the throat and

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FIG. 1. Carotid arteriogram demonstrating carotid artery pushed forward by an extrinsic mass.

FIG. 2. Film taken one second later showing the exceedingly vascular tumor lying beneath the mastoid process and under the mandible.

how fortunate the doctor was who had removed the tissue for biopsy in the area had recovered only tonsillar tissue, and not the wall of the carotid artery (figs. 2 and 3).

Removal of the tumor was attempted. A hard, exceedingly vascular, seemingly encapsulated mass was exposed high in the neck under the mandible. It could be

FIG. 3. Anteroposterior projection indicating the tangle of blood vessels supplying the tumor are lateral to the internal carotid artery.

separated from the carotid artery without difficulty. The vagus and spinal accessory nerves formed an integral part of the mass. The hypoglossal nerve also was surrounded by the tumor but it did not appear to be invaded by it. The jugular vein was ligated to get exposure. Since the mass was so large, an attempt was made to scallop it with the electrocautery. Bleeding was so brisk that despite the coagulation of several feeding arteries, this procedure had to be abandoned. Tissue for biopsy was taken and because the pathologist who examined the frozen section reported a malignant tumor, further operation was abandoned.

Permanent sections secured a few days later disclosed a typical nonchromaffin paraganglioma. Several days were allowed for the edema in the neck to subside. During this time hoarseness was first apparent due to injury to the vagus nerve. Also paralysis and atrophy of the tongue was evident since the twelfth nerve had been cut.

Reoperation was undertaken a week after the first attempt. The carotid artery and muscles of the neck were separated from the tumor, but the neoplasm remained firmly attached to the base of the skull. Visual exposure could not be obtained but digital palpation indicated that the mass was being held by string-like structures, probably the nerves of the jugular foramen. To remove the mass the operator's fingers were slipped over the cephalad portion and it was drawn out. The jugular vein was broken at the foramen and the ninth, tenth and eleventh nerves were avulsed. A piece of oxycel gauze was slipped into the jugular vein, which controlled the bleeding. The tumor bed could be examined carefully and there was no evidence of intracranial extension. The jugular fora-

FIG. 4. Neoplasm as removed at operation. It decreased somewhat in size when separated from its profuse vascular supply. Defects in lower end of specimen indicate unsuccessful attempt at first operation to reduce size by use of the electrosurgical loop. Arrow points to the cephalid portion of the vagus nerve which was anchored out of the jugular foramen.

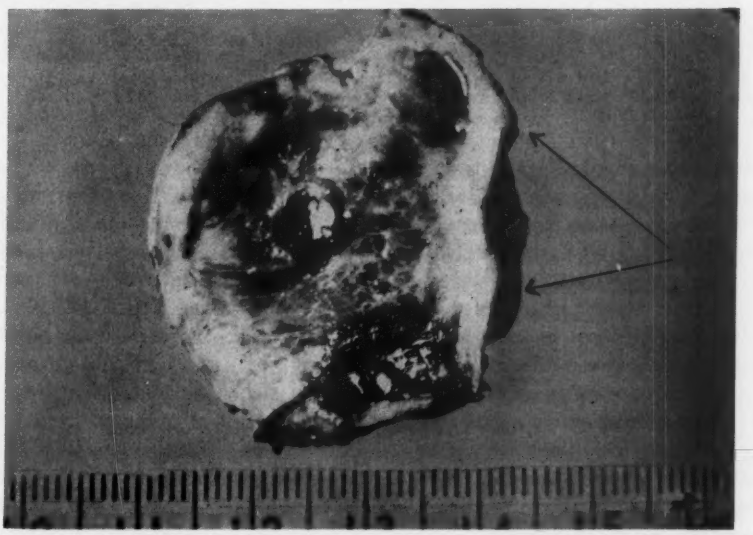

FIG. 5. Longitudinal section through tumor and vagus nerve. Note intimate relationship between neoplasm and nerve. The ninth, eleventh and twelfth nerves were easily extracted from the tumor mass, and their sheaths were not invaded by the neoplastic tissue. Arrows point to nerve.

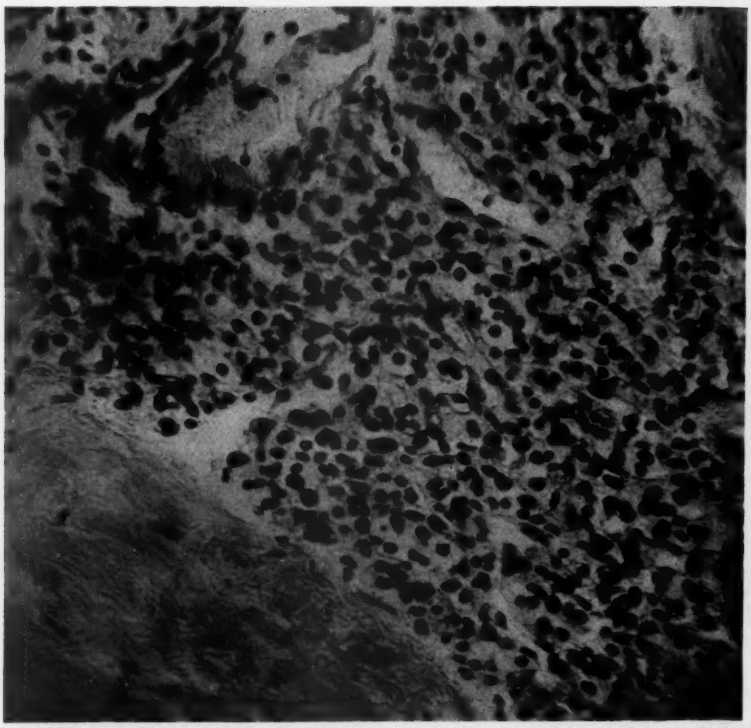

FIG. 6. Microscopic section showing that the tumor is composed of small epithelial cells containing deeply staining nuclei and moderate amounts of cytoplasm. The neoplastic cells can be seen invading between the fibers of the vagus nerve.

men was not enlarged. The tumor was covered with a smooth capsule. It appeared to be arising from the vagus nerve. Close inspection of the proximal end of the vagus nerve seemed to rule out an intracranial extension and it appeared that the tumor was entirely in the cervical region. The ninth and eleventh nerve fibers while incorporated within the tumor mass, did not appear to be invaded by it. Microscopic pictures were identical with those of the biopsy (figs. 4, 5, 6).

The postoperative course was uncomplicated. Besides hoarseness and atrophy of the tongue, the patient had quite a difficult time learning to swallow again. Due to the anesthesia in the posterior portion of the tongue and pharynx on the right as well as laxness of muscle tone, food frequently found its way into the upper trachea. Violent coughing resulted which was quite distressing. At the end of 10 days, however, the patient was eating sufficient food. The usual disability of poor shoulder stabilization on the right also was present, due to paralysis of the trapezius muscles.

For the past year, the patient has remained well. There is no evidence of recurrence or metastasis. Care must be taken in swallowing foods, but water, liquids and solids can be swallowed if done slowly. There are no complaints

about the shoulder now (it is fortunate that the patient is not required to do much work) and the atrophy of the tongue is unnoticed. The adjustment to the unilateral loss of the ninth, tenth, eleventh and twelfth cranial nerves has been remarkable.

DISCUSSION

While nonchromaffin paragangliomas are unusual tumors, those arising from the intravagal ganglion are very rare indeed. We have been able to discover only 1 other case that has been amenable to surgical attack.

It seems clear that cells of the nonchromaffin type may be located within the vagus nerve, in close association with both jugular ganglion and ganglion nodosum. Portions of the latter ganglion are extracranial and it should be possible to excise tumors arising from this region.

Neoplasms arising from the intracranial portion of the ganglia have been reported somewhat more frequently. Albernaz and Bucy[1] described such a case and cite a few other examples gleaned from the medical literature. Several reports of tumors of the glomus jugularis recently have appeared but these arise from a structure separate from the vagus nerve and, while they may invade the region of the jugular foramen, they should not be confused with intrinsic neoplasms of the vagus nerve.

Identification of nonchromaffin cells within the normal vagus is not always possible. The pathologic laboratory of this clinic recently has studied the vagi from 3 normal individuals and were unable to demonstrate with certainty such cells in the neighborhood of the ganglion nodosum. While the standard gross anatomic texts all show the cervical portion of the nodose ganglion, little has been said about its histologic structure. It seems established, however, that at least in some individuals collections of these nonchromaffin cells are present.

Tumors arising from these structures are difficult to diagnose clinically. They are deep in the structures of the neck, and palpation is almost impossible due to the overlying mandible and sternocleidomastoid muscle. Intraoral palpation is easier, as noted by Stout[3] as well as in our patient.

Carotid angiography was exceedingly important as an adjunct in the surgical diagnosis. First, it dismissed the clinical impression of an aneurysm. Second, it disclosed a circumscribed, highly vascular tumor that lay just beneath the mastoid process and pushed the internal carotid artery forward. The large tangle of blood vessels suggested a preoperative diagnosis of a carotid body tumor. Finally it seemed to exclude an intracranial extension. The only other instance of which we are aware in which angiography was used in the evaluation of a nonchromaffin tumor is the case reported by Alexander and Adams.[2]

Attempts at surgical excision were abandoned at the first operation when the pathologist reported that the tumor was malignant, even though on gross inspection it appeared benign. When its true nature was established, operation was resorted to once more, even though we knew that the tenth, eleventh and twelfth cranial nerves would have to be sacrificed. Albernaz and Bucy pointed out that this might result in a grave neurologic deficit, but they felt justified in attempting it.

Vernet's and Villaret's syndromes are well known and such patients are not too severely incapacitated. Occasionally the nerves of the jugular foramen are damaged during posterior fossa surgery with similar results. The patient described above goes through her daily routine with very little trouble. Since she has learned to swallow and eat carefully, she has surprisingly little disability.

The efficacy of radiation therapy in the treatment of nonchromaffin tumors still is open to much debate. Most authors state emphatically it is of no use. The patient treated by Alexander and Adams and other isolated instances seems to cast doubt on this belief. Dependence on this form of therapy when the tumor is in the intracranial portion of the vagus nerves seems to be almost a necessity, but those appearing in the neck may be cured by operation.

SUMMARY

The second case of a nonchromaffin paraganglioma arising in the cervical portion of the vagus nerve is reported. Surgical excision was possible although the approach was different to that reported in the first case reported by Stout. Despite the resection of the ninth, tenth, eleventh and twelfth cranial nerves, the clinical course of the patient has been very satisfactory.

REFERENCES

1. Albernaz, J. G., and Bucy, P. C.: Non-chromaffin paraganglioma of jugular foramen, J. Neurosurg. *10:* 663 (Nov.) 1953.
2. Alexander, E., and Adams, S.: Tumor of glomus juglare, J. Neurosurg. *10:* 672 (Nov.) 1953.
3. Stout, A. P.: Malignant tumors of peripheral nerves, Am. J. Cancer *25:* 1 (Sept.) 1935.

THE IMPORTANCE OF MEDIASTINAL METASTASES IN CARCINOMA OF THE THYROID

Mediastinal metastases from carcinoma in the thyroid gland are receiving increased attention from surgeons who desire to fulfill the en bloc principle of excising a cancer bearing organ and its regional lymphatic drainage. Although known to anatomists for well over a century, the mediastinal portion of the lymphatics draining the thyroid gland only recently has been incorporated in radical procedures designed to remove malignant disease arising in this organ. Technical advances in anaesthesia and resuscitation make practical transsternal anterior mediastinal and radical neck dissection of the lymphatics combined with excision en bloc of the entire cancer bearing organ.

Cancer of the thyroid gland characterized by a papillary form of architecture is increasing in frequency. Both here and abroad reports indicate that this neoplasm has increased from approximately 15 per cent of the cases to as much as 80 per cent. In the writer's series the incidence of papillary cancer has risen from about 33 per cent prior to 1945 to 50 per cent thereafter. Since it is generally agreed that the papillary forms of thyroid carcinoma have a predilection to enter lymphatics, the importance of the mediastinal portion from the thyroid is evident. In their 12 reported and 4 as yet unrecorded cases, McClintock and his associates have found mediastinal metastases in 11.

It is fortunate indeed that the cancer showing an increased frequency in the thyroid gland is one that metastasizes via the lymphatics. Its tendency to remain localized for long periods in the nodes draining the thyroid affords the surgeon an opportunity to apply Halsted's en bloc principle of excision. To gain this objective when dealing with lesions of the thyroid gland the surgeon must be aware of the importance of the anterior mediastinum.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Urology. MEREDITH CAMPBELL, M.D., F.A.C.S., Emeritus Professor of Urology, New York University with the collaboration of 51 contributing authorities. Three volumes, 2,356 pages, 1,148 figures. Publisher: W. B. Saunders Co., Philadelphia, 1954. Price: $60.00

Many excellent textbooks of urology have been published since 1900 when urology became an accepted surgical specialty, and this new study edited by Dr. Campbell is one of the best. In 1926 Saunders published a similar set of volumes edited by the late Dr. Hugh Young which for many years was a standard textbook in urology. Twenty-eight years later this new text written by competent authors again will become a definitive reference text in urology. The collaborating authors are all outstanding and their contributions bring up-to-date the many developments of the last few years. An excellent integration with the basic sciences is achieved by the use of monographs by Doctors Pitts, Huggins, Moore, Ash, Engle, Hotchkiss, Jenkins and others. Related fields are also covered by authorities. The field of urology has been greatly enriched by this splendid effort.

WILLIAM L. VALK, M.D.

Retropubic Prostatectomy for Benign Enlargement of the Prostate Gland. By FRANCIS A. BENEVENTI, M.D., F.A.C.S., Attending Urologist, Oswald Swinney Lowsley Foundation, St. Clare's Hospital; Attending Urologist, Lincoln Hospital; Associate Attending Urologist, New York Polyclinic Hospital and Medical School; Assistant Attending Urologist, James Buchanan Brady Foundation of the New York Hospital, New York, New York. Art Direction and 44 original drawings, By WILLIAM P. DIDUSCH. Charles C Thomas, Publisher, Springfield, Illinois. Price: about $14.00.

Retropubic prostatectomy has been performed many thousand times since Mr. Terence Millin reintroduced this old operation in this country in 1945. This monograph is very well written and superbly illustrated and is an excellent addition to the few previous studies of this operation. The chapters on preoperative and postoperative care of the patient are well done and the description of the technic of operation is excellent. The advantages and disadvantages of retropubic prostatectomy are discussed in relation to the author's own series of 100 consecutive operations and they are fairly reported. A helpful bibliography follows each chapter and an interesting history of open prostatectomy completes the volume. Retropubic prostatectomy is an accepted surgical procedure and this monograph will be welcomed by all who use this operation.

WILLIAM L. VALK, M.D.